

SECOND EDITION

Anesthesia

EMERGENCIES



EDITED BY

KEITH J. RUSKIN

STANLEY H. ROSENBAUM

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Anesthesia Emergencies

Second Edition

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Contents

Preface [vii](#)

Contributors [ix](#)

1	Crisis Resource Management: Before the Emergency	
	<i>Yili Huang and Viji Kurup</i>	1
2	Airway Emergencies	
	<i>Laebe Lester and Lauren Berkow</i>	7
3	Respiratory Emergencies	
	<i>Vivek K. Moitra and Tricia E. Brentjens</i>	35
4	Cardiovascular Emergencies	
	<i>Ajoy Katari and Benjamin Kohl</i>	59
5	Thoracic Emergencies	
	<i>Peter S. Burrage, Marc S. Azran, and Michael Nurok</i>	99
6	Metabolic and Endocrine Emergencies	
	<i>Greta L. Piper and Adrian A. Maung</i>	125
7	Neurosurgical and Neurologic Emergencies	
	<i>Kellie A. Park</i>	173
8	Obstetric Emergencies	
	<i>Robert R. Gaiser</i>	191
9	Pediatric Emergencies	
	<i>Anna Clebone and Bradley Besson</i>	243
10	Miscellaneous Problems	
	<i>Sara E. Neves and Keith J. Ruskin</i>	281
11	Surgical Emergencies	
	<i>Maria D. Georgiades, Stephen M. Luczycki, and Linda L. Maerz</i>	301

12	Postanesthesia Care Unit <i>Sean M. Quinn and Keith A. Candiotti</i>	329
13	Regional Anesthesia Complications <i>Nikhil Bhatnagar</i>	357
14	Equipment Problems <i>René R.P.M. Hagenouw and James B. Eisenkraft</i>	375
15	Procedures <i>Trevor Banack, Ann Mellookaran, and William Rosenblatt</i>	393
16	Disaster Management <i>Michael J. Murray</i>	421
17	Ethical Considerations <i>Robert B. Schoenberger and Stanley H. Rosenbaum</i>	441

	Index	449
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Preface

Anesthesiology is unique among medical specialties because most anesthetics and surgical procedures are uneventful. Critical events can, however, occur without warning, are usually sudden, and may be life-threatening. Anesthesiologists must be ready to detect and manage unpredicted emergencies at any time. Although anesthesia itself has a low risk (independent of patient disease), a recent review offered suggestions as to how safety in the perioperative period can be further improved and how postoperative complications can be avoided.¹

The initial response to a critical event may determine its outcome. The use of checklists and established protocols, long an accepted practice in aviation, can help health care providers quickly establish a diagnosis and begin treatment. *Anesthesia Emergencies* is written to help an anesthesia provider deal with common complications or unforeseen emergencies that happen during the perioperative period.

This updated edition of *Anesthesia Emergencies* can be used to prepare for emergencies that may occur in the future, and to deal with critical events as they happen. Chapters have been organized alphabetically, and each section within a chapter is arranged alphabetically by type of problem. Critical information has been highlighted. Each chapter contains one or more references to textbook chapters or review articles that will provide additional information. The second edition has been expanded to include chapters on crisis management and disaster preparedness, and new emergencies have been added to nearly every chapter.

Health care providers must have the knowledge, skills, and equipment necessary to resolve unforeseen emergencies. This book will ideally be used to prepare for emergencies before they occur. The authors recommend that if a specific type of problem can be anticipated (e.g., massive hemorrhage in a postpartum patient being brought to the operating room) the appropriate chapter should be reviewed beforehand. All personnel on the care team should be briefed on the critical events most likely to occur, and the actions that will be taken during an emergency. This book can be used outside of the operating room to prepare for future events

1 Wacker J, Staender S. The role of the anesthesiologist in perioperative patient safety. *Curr Opin Anaesthesiol*. 2014 Dec 27(6):649–656.

by reviewing a section and thinking through the steps that would be needed for successful resolution of the problem.

This second edition will ideally be one part of an organized approach that includes simulation and training in crisis resource management. The authors recommend that when an emergency occurs, the anesthesia provider call for help as quickly as possible and delegate tasks as personnel arrive. There should be one person who is clearly in charge and directs the others, but that person should ask for advice and help as needed. When an anticipated critical event occurs, the Immediate Management, Differential Diagnosis, and Subsequent Management sections can be used as checklists, to help the anesthesia provider remember each of the steps that must be taken. If an unforeseen emergency occurs, the same sections can be used as a Do list. Follow the suggested procedure, and then refer to the article in Further Reading for more information after the patient has been stabilized.

This book would not have been possible without the help of many people. The authors would first like to thank their families for their constant support. We would like to thank our editors, Andrea Knobloch and Rebecca Suzan, for their advice and guidance. We also thank our authors, who produced outstanding manuscripts and turned them in on time. We acknowledge Keith Allman, Andrew McIndoe, and Iain Wilson, whose Oxford Publication, *Emergencies in Anaesthesia*, served as inspiration for this work. Lastly, we thank the residents and faculty of the Yale University School of Medicine, Department of Anesthesiology, for their critical review of the manuscript and their thoughtful comments.

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Chapter 1

Crisis Resource Management: Before the Emergency

Yili Huang and Viji Kurup

Introduction to Crisis Resource Management	2
Key Principles	2
Checklists	4
Training	5
Future Directions	5

Introduction to Crisis Resource Management

Emergencies in the operating room are frequently associated with an uncertain diagnosis and limited access to diagnostic interventions and are managed by members of a multidisciplinary team. Inefficient teamwork and errors in decision making can rapidly exacerbate the situation and possibly lead to fatal errors.

Crisis resource management (CRM) focuses on development of nontechnical skills such as decision making, interpersonal behavior, and team training in order to facilitate effective teamwork in a crisis situation. Crisis resource management aims to improve performance and decrease errors of complex tasks at the individual and team levels.

Key Principles

The guiding principle of CRM is that during a crisis, medical knowledge is required but not sufficient for a successful outcome. Management of the complex interactions among the environment, equipment, and different care teams is essential to function effectively in a crisis situation. These principles (Box 1.1) include an understanding of the roles of leaders and team members; rapid,

Box 1.1 Key Principles

- Leader
 - First to arrive or knows the most about the patient
 - Steps back, gets the big picture
 - Does not become involved in physically doing any task
 - Organizes the team and delegates responsibility
 - Constantly assimilates information from team members
- Team member
 - Assumes assigned task
 - Not inferior but person best suited for specific task
 - Provides constant open discussion and updates with the leader
 - Centralizes all communication through the leader and does not communicate directly with other members in order to maximize team awareness
 - Does not usurp the role of team manager from the leader

Box 1.1 (continued)

- Communication
 - Introduce yourself
 - Address people directly
 - Make eye contact
 - Employ close loop communication by repeating back orders as a method to ensure that they have been heard correctly and are acknowledged
 - Use nonjudgmental comments
- Assessment
 - Leaders should constantly step back and reassess situation that may be evolving
 - It is important to be aware of and avoid fixation errors
 - Verbal reviews of the situation are important to provide a shared mental model and question the management plan when appropriate
- Resources
 - Prepare for anticipated needs
 - Promote outside the box thinking
 - Constantly assess the available resources and make full use of them
- Support
 - Asking for help is a sign of maturity, not weakness
 - Understand that outside help may be called
 - Know when to, whom to, and what type of help to call

From Murray WB. Crisis resource management among strangers: principles of organizing a multidisciplinary group for crisis resource management. *J Clin Anesthesiol.* 2000; 12(8): 634.

effective communication; situational awareness; and knowledge of available ancillary resources.

In a crisis, the leader does not become involved in any physical tasks, but instead takes a step back to get the big picture by assimilating information from team members, organizes it, and delegates the appropriate responsibility. The leader is often the first person to arrive at the scene, but may later be replaced by the person who knows the most about the patient. The leadership role may be transferred between providers if, for example, the patient's primary physician arrives or the team leader has expertise that is required (e.g., airway management).

Team members are responsible for completing tasks assigned by the team leader. All communication between team members

should go through the team leader, as should any updates or insights into management of the crisis. This will allow the leader to coordinate the activities of everyone involved in the event and maximize team awareness.

Effective communication is vital to successful crisis management. Not all providers may be familiar with each other, so introductions between team members may be beneficial if time permits. Introductions should include not only the person's name, but also his or her role in patient management. For example, "My name is Yili Huang; I am an anesthesiologist." *Closed loop communication* involves repeating back instructions. If, for example, the team leader instructs Dr. Huang to manage the airway, he should then reply, "I'll manage the airway." This lets the leader know that his instructions have been heard and understood, and also allows him or her to correct an erroneous instruction. Maintaining eye contact while addressing other team members directly and employing nonjudgmental comments all help make communication effective during a crisis.

Constant assessment of the crisis situation is an important part of CRM. Leaders should repeatedly take a step back to analyze the big picture. Verbal review of the situation is also important to help provide a shared mental model for the team. This helps prevent fixation errors and allow critique of the management plan when appropriate. Throughout the event, the team should prepare for anticipated needs by constantly reviewing available resources and fully utilizing them. Any member of the team can request assistance if necessary, and one of the responsibilities of the team leader is to continually be aware of what help is available and what type of support should be summoned.

Checklists

Checklists have become a vital part of CRM, and have been shown to aid performance in rare and unpredictable crises. Checklists are beneficial because the stress during crisis can impede the team's ability to recall critical steps in management. Checklists provide relevant, vital information packaged in a visually striking presentation that can be quickly and easily accessed. Checklists have been shown to improve teamwork, standardization, and performance during simulated critical events in the operating room. The current book is in reality a series of checklists for emergency situations. Therefore, this book could be used as a cognitive aid in crisis scenarios.

Box 1.2 includes three of the most accessible and validated checklists currently available.

Box 1.2 Available Crisis Checklists

- American Heart Association Basic and Advanced Cardiac Life Support
 - www.heart.org
- Project Check OR Crisis Checklist
 - www.projectcheck.org
- Emergency Manual
 - Emergencymanual.stanford.edu

Training

Both initial and recurrent training are important and critical to successful CRM because of the diverse background of the operating room team. The goal is to ensure that the team functions as a unit during critical events. No matter the type of training, the team needs to adopt a willingness to cooperate toward the common goal of patient health and safety.

Future Directions

Petrosoniak and colleagues proposed the “triple threat” framework in a recent publication (Box 1.3). The triple threat encourages the creation of a shared mental model in which team members can train to anticipate each others’ actions by understanding each others’ perspectives and needs. Stress inoculation training identifies that stress impairs team performance, and effective management of this stress can allow for improved team function. Last, it recognizes that time pressures and the demand for high-stakes

Box 1.3 Triple Threat Model

- Creation of shared mental models
 - Developed by combat aviation
 - Team behavior is best when members can predict each others’ needs
 - Cross-training—team members assume one another’s position to understand each other’s roles
 - Interdisciplinary team training helps develop shared understanding of team

Box 1.3 (continued)

- Stress inoculation training
 - Borrowed from military flight teams
 - Acute stress impairs team performance
 - Stress inoculation training helps desensitize, identify, and manage acute stress
- Combat cognitive error and improve decision making
 - Pressures during crisis promote cognitive error
 - Cognitive de-biasing require understanding and reflecting one's thought process to avoid cognitive errors

From Petrosniak A, Hicks CM. Beyond crisis resource management: new frontiers in human factors training for acute care medicine. *Curr Opin Anesthesiol.* 2013; 26: 699–706.

decisions promote cognitive errors, and it is necessary to learn the techniques of metacognition and thereby improve decision making.

Further Reading

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Chapter 2

Airway Emergencies

Laeben Lester and Lauren Berkow

Airway Fire	8
Aspiration	9
Surgery- and Anesthesia-Related	10
Bleeding following Tonsillectomy	13
Cannot Intubate/Can Ventilate	15
Cannot Intubate/Cannot Ventilate	16
Difficult Mask Ventilation	18
Difficult Ventilation through an Endotracheal Tube	18
Hemoptysis	21
High-Risk Tracheal Extubation	24
Intrinsic Upper Airway Obstruction	27
Laryngospasm	29
Ludwig's Angina	30
Rapid-Sequence Intubation	32

Airway Fire

Definition

Ignition of combustible materials in the airway.

Presentation

Any fire requires three components: a fuel source, a source of ignition, and oxygen. In the operating room, alcohol-based skin prep solutions, drapes, and the plastic endotracheal tube are fuel sources, cautery and laser beams are the primary sources of ignition, and oxygen is often enriched.

Immediate Management

- Immediately disconnect the breathing circuit from the endotracheal tube.
- Inform the surgeons that a fire has occurred.
- Remove the endotracheal tube.
- Stop flow of all airway gases, especially N_2O .
- Remove all other flammable materials from the airway.
- Pour saline into the airway to extinguish any flaming debris.
- Reintubate the patient, even if injury is not immediately apparent.

Subsequent Management

- After an airway fire has occurred, the patient should be reintubated and examined by fiberoptic bronchoscopy to determine the extent of airway injury and remove any residual debris.
- Admission to an intensive care unit (ICU) is often required.

Prevention

- Use the lowest FiO_2 that the patient will tolerate.
- Determine whether there is risk of a surgical fire before every procedure and formulate a plan of action that will be taken if a fire occurs.
- Display a protocol for fire prevention and management in every operating room.
- Whenever the surgical site is near the airway, a 60-mL syringe filled with saline solution should be immediately available.
- Ensure that the surgeon does not enter the trachea with electrocautery during a tracheostomy.
- When using lasers on the airway, use the lowest possible FiO_2 (40% or less).
- Avoid use of nitrous oxide for surgery near the airway.

- Consider insufflating the endotracheal tube (ETT cuff) with saline rather than air.
- Consider securing the airway as opposed to using a face mask or nasal cannula to administer O₂ during head and neck procedures.

Special Considerations

- The only indication of an airway fire may be a puff of smoke and a flash of light.
- Alcohol-based skin cleansing solutions are highly flammable, and vapors can be trapped under the drapes if the site is not completely dry.
- Alcohol-based solutions should not be allowed to pool, and should be given 3 minutes to dry before applying surgical drapes.
- Fires caused by alcohol-based prep solutions may be invisible under ordinary room lighting and can spread within seconds.
- If a face mask or nasal cannula oxygen is being used to deliver O₂, it may accumulate under drapes and exacerbate a fire.

Further Reading

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Aspiration

Definition

Passage of liquid or particulate material into the airway below the vocal cords.

Risk Factors for Aspiration with General Anesthesia

Patient-Related

- Emergency surgical procedures
- Inability to clear pharyngeal secretions/poor or absent gag reflex
 - Altered mental status
 - Head injury
 - Medications

Risk Factors for Aspiration with General Anesthesia (*continued*)

- Bulbar palsy
- Multiple sclerosis
- Parkinson disease
- Guillain-Barré syndrome
- Muscular dystrophies
- Cerebral palsy
- Increased volume of food and acid in the stomach
 - Recent ingestion of food or fluids
 - Poor gastric emptying/gastroparesis
 - Obesity
 - Diabetes
 - Autonomic neuropathy
 - Pregnancy
 - Ileus
 - Renal failure
 - Head injuries
 - Pain
 - Stress
 - Trauma
- Intestinal obstruction
 - Pyloric stenosis
- Incompetence of the lower esophageal sphincter
 - Esophageal reflux
 - Achalasia cardia
 - Hiatal hernia
- Increased intra-abdominal pressure
 - Obesity
 - Pregnancy
- Patients with a history of prior upper abdominal surgery
- Extremes of age

Surgery- and Anesthesia-Related

- Poor induction technique
 - Large tidal volume or high airway pressure during mask ventilation
 - Incorrectly performed cricoid pressure

- Difficult intubation resulting in:
 - Gas insufflation into the stomach during mask ventilation
 - Inability to intubate and protect the airway with a cuffed tube
- Medications and drugs
 - Opioids
 - Topicalization of the airway leading to suppression of the gag reflex
- Depth of anesthesia
 - Manipulation of upper airway under light anesthesia, leading to gagging and vomiting
- Patient positioning
 - Trendelenburg position
 - Lithotomy position
- Increased intra-abdominal pressure
 - Intra-abdominal air insufflation
 - External pressure on the abdomen

Presentation

- Material found in the oropharynx
- Wheezing
- Elevated airway pressure
- Infiltrates seen on chest X-ray
- Clinical findings of acute respiratory distress syndrome as syndrome develops

Pathophysiology

Factors affecting outcome after aspiration include:

- pH of aspirate
 - In general, the extent of injury increases with the acidity of the aspirated material.
 - Aspiration of bile is associated with extensive tissue injury.
- Volume of aspirated material
- Particulate matter
 - Particulate matter increases the mortality and incidence of pneumonitis and bacterial overgrowth.
- Bacterial load
- Blood in the airway
 - Blood in the airway generally produces minimal injury, but may predispose the patient to infection.
- Host responses

DIFFERENTIAL DIAGNOSIS

- Laryngospasm or airway obstruction during intubation
- Bronchospasm, wheezing, or crackles following intubation
- Hypoventilation, dyspnea, apnea
- Reduced pulmonary compliance (acute respiratory distress syndrome [ARDS])

Immediate Management

- Increase FiO_2 to 100%.
- Position the patient with the head down.
- Maintain cricoid pressure. (Pressure must be released during active vomiting to avoid esophageal rupture.)
- Suction the nasopharynx and oropharynx.
- Intubate the trachea. (Consider rapid-sequence induction if the patient is not cooperative.)
- Suction the lower airway.
- Initiate mechanical ventilation as indicated. (Positive end-expiratory pressure [PEEP] of at least 5 cm H_2O may be required.)
- Administer bronchodilators (e.g., nebulized albuterol) as indicated.
- Perform bronchoscopy and remove particulate matter.
- Defer planned or noncritical surgery where feasible.
- Obtain chest X-ray and arterial blood gas as indicated.

Subsequent Management

- Do not administer routine prophylactic steroids.
- Do not start empiric antibiotics unless there is a clear risk factor (e.g., aspiration of feculent matter). In most cases, initiate antibiotic therapy when there is a clear diagnosis of pneumonia.
- Do not administer H_2 blockers or proton pump inhibitors. Antacids and prokinetic drugs have not been shown to improve outcome after aspiration.
- Ensure careful fluid management (because volume shifts may occur that lead to pulmonary edema).

Prevention

- Follow nil per os (NPO) guidelines, except for urgent or emergency surgery.
- Ensure early control of the airway in patients with poor gag reflex or altered sensorium.
- Exercise increased vigilance during intubation and extubation.

- If indicated, administer H_2 blockers or proton pump inhibitors at least 90–120 minutes prior to the surgical procedure.
- Use nonparticulate antacids (e.g., sodium citrate) that decrease the gastric pH.
- Administration of prokinetic agents (e.g., metoclopramide) before surgery may decrease the risk of aspiration by decreasing the volume of gastric contents.
- Use rapid-sequence intubation where appropriate.
- Consider an awake intubation technique if difficulty is suspected.

Special Considerations

- The incidence of aspiration in adults is roughly 1 in 3000 anesthetics. In patients undergoing emergency surgery, this risk increases to 1 in 600–800, and for caesarean sections under general anesthesia the incidence is 1 in 400–900.
- Children are at increased risk for aspiration, with an overall incidence of 1 in 2600 and an incidence of 1 in 400 cases during emergency surgery.
- The consequences of aspiration can be catastrophic: Patients requiring ventilation for more than 48 hours postaspiration have a 50% mortality rate.

Further Reading

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Bleeding following Tonsillectomy

Definition

Significant bleeding from the surgical field after tonsillectomy surgery.

Presentation

Post-tonsillectomy bleeding is most common in the first 24 hours, but can occur within the first week or longer after surgery. Signs of

bleeding can include blood coming from the mouth or nose, spitting bright red blood, vomiting bright red or old blood (“coffee ground emesis”), or a metallic taste in the mouth.

Pathophysiology

Causes of bleeding after tonsillectomy include incomplete surgical hemostasis, treatment with antiplatelet agents or anticoagulants, and various coagulopathic states (e.g., hemophilia, von Willebrand disease).

DIFFERENTIAL DIAGNOSIS

- Hemoptysis
- Nontonsillar bleeding

Immediate Management

- Evaluate the patient’s airway rapidly.
- Administer 100% oxygen.
- Request an emergency evaluation by the otolaryngology service.
- Consider reintubation if the patient is bleeding rapidly or is unable to protect his or her airway.
- If intubation is necessary, consider moving the patient to the operating room if this is a safe option.
- Ensure that adequate suction is available. Blood in the oropharynx may obscure the view during laryngoscopy.
- Assume that the patient has a full stomach and is at risk for extensive pulmonary aspiration. Rapid-sequence induction of general anesthesia is recommended.
- Consider a “double set-up” approach with surgeons prepped and gowned for surgical airway before induction of anesthesia.
- Restore intravascular volume if the patient has signs of hypovolemia.
- Consider surgical re-exploration of the tonsillar bed.

Special Considerations

- The blood supply to the tonsils comes from the external carotid artery and its branches. It is sometimes necessary to embolize or ligate the external carotid artery in patients with severe hemorrhage.

Further Reading

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Cannot Intubate/Can Ventilate

Definition

Inability to intubate the trachea. Adequate face mask ventilation is still possible.

Immediate Management

- Call for help.
- Ensure that the FiO_2 is 100%.
- Consider inserting a supraglottic airway (SGA) (e.g., a laryngeal mask airway) if appropriate.
- If insertion of an SGA is not feasible, and if another attempt at intubation is not appropriate, awaken the patient. Consider deferring the surgery or proceeding with awake intubation.
- If ventilation becomes difficult, proceed to Cannot Intubate/ Cannot Ventilate.
- If the patient becomes hypoxic and cannot be ventilated, consider a surgical airway (i.e., cricothyrotomy).
- If another attempt at intubation is warranted, consider the options listed in Box 2.1.
- Refer to the ASA Difficult Airway Algorithm in the inside front cover of this book.

15

Box 2.1 When an Initial Attempt at Intubation Fails

Place the patient's head in an optimal position (e.g., head in “sniffing position,” use of pillows or towels to “ramp up” obese patients—optimal position is achieved when the tragus of the ear is level with the sternal notch).

Consider one or more of the following options:

- External laryngeal manipulation
- Consider asleep fiberoptic intubation. (This should be attempted early because blood and secretions make this procedure more difficult.)
- Consider an alternate laryngoscope blade (e.g., larger or smaller size, straight instead of curved).

(continued)

Box 2.1 (continued)

- Consider a video laryngoscope.
- Consider a gum elastic bougie.
- Intubation through an SGA.

As a general rule, awaken the patient after three unsuccessful attempts and proceed with intubation using an FOB technique or other method (e.g., retrograde intubation technique) while the patient breathes spontaneously.

Subsequent Management

- Document the intraoperative events carefully in the medical record, with special attention to those techniques that were successful.
- Explain the sequence of events to the patient and advise him or her to warn future anesthesia providers.
- Write the patient a “difficult airway” letter. Advise the patient to enroll in a medical registry such as MedicAlert.

Further Reading

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Cannot Intubate/Cannot Ventilate**Definition**

Inability to intubate the trachea. Inability to ventilate the patient by face mask.

Immediate Management

- Call for help. A surgical airway may become necessary.
- Ensure that the FiO_2 is 100%.
- Call for the difficult airway cart.
- Reposition the patient's head and jaw.
- Treat suspected laryngospasm with propofol 0.25–0.8 mg/kg or succinylcholine 0.1–2 mg/kg.

Immediate Management (continued)

- Insert an airway (e.g., oropharyngeal, nasopharyngeal, laryngeal mask airway) In many such cases the airway can be rescued with insertion of a supraglottic airway.
- Consider two-person ventilation. One person holds the mask in position and delivers a jaw thrust using both hands while the other ventilates the patient by hand using the reservoir bag and the emergency O₂ flush valve as needed.
- If oxygenation remains satisfactory, consider the use of a video laryngoscope or fiberoptic intubation.
- Consider a surgical airway (e.g., cricothyrotomy) or transtracheal jet ventilation if the patient is hypoxic.
- Refer to the ASA Difficult Airway Algorithm in the inside front cover of this book.

Subsequent Management

- Document the intraoperative events carefully in the medical record, with special attention to those techniques that were successful.
- Explain the sequence of events to the patient and advise him or her to alert future anesthesia providers.
- Write the patient a “difficult airway” letter and advise the patient to enroll in a medical registry such as MedicAlert.
- If a surgical airway has been attempted, request an emergency ear-nose-throat (ENT) consultation.

Special Considerations

- Ensure that extra help is available before attempting to manage the airway if difficult ventilation and/or ventilation is predicted. Be certain that the patient is adequately preoxygenated before inducing anesthesia. Optimize patient position (“sniffing position,” use of pillows or towels to “ramp up” obese patients). Have special intubation equipment readily at hand.

Further Reading

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Difficult Mask Ventilation

Definition

Inability to provide adequate ventilation with a face mask due to inadequate mask seal, excessive gas leak, or excessive resistance to gas flow.

Presentation

Absent or inadequate chest movement or breath sounds, signs of airway obstruction, cyanosis, gastric air entry causing dilatation, hypoxemia, absent or inadequate exhaled CO₂, or low gas flows (reservoir bag does not fill).

Pathophysiology

There are five independent predictors for difficult face mask ventilation: (1) age >55 years; (2) body mass index (BMI) >26 kg/m²; (3) presence of a beard; (4) edentulous patient; and (5) history of snoring.

Immediate Management

- Increase FiO₂ to 100%.
- Administer a jaw thrust.
- Ensure that the face mask is correctly sized.
- Consider an oral airway or a nasopharyngeal airway.
- Consider inserting a supraglottic airway.
- Consider two-person ventilation, with one person using both hands to get a good face mask fit and the second person doing the ventilation.
- Consider endotracheal intubation.

Further Reading

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Langeron O, Masso E, Huraux C, Guggiari M, Bianchi A, Coriat P, Riou B. Prediction of difficult mask ventilation. *Anesthesiology*. 2000; 92: 1229–1236.

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Difficult Ventilation through an Endotracheal Tube

Definition

High airway pressure and/or inability to adequately ventilate with a tracheal tube in place.

Presentation May Include

- Stiff rebreathing bag during manual ventilation
- Increased airway pressure
- Hypercarbia/elevated end-tidal CO₂ levels
- Unilateral breath sounds
- Atelectasis seen on chest X-ray

DIFFERENTIAL DIAGNOSIS

- Endobronchial intubation
- Bronchospasm
- Kinked endotracheal tube
- Blood or secretions occluding endotracheal tube
- Pneumothorax
- Anaphylaxis
- Other causes (e.g., ARDS, pulmonary edema)
- Light anesthesia
- Patient/ventilator dyssynchrony

Immediate Management

- Administer 100% oxygen.
- Auscultate the lung fields for wheezes, crackles, and unilateral breath sounds (endobronchial intubation).
- Pass a suction catheter down ETT to rule out kinking or the presence of blood, secretions, or a mucous plug.
- Ventilate the patient with a self-inflating bag (Ambu Bag, Ambu Corp., Ballerup, Denmark) to rule out a problem with the anesthesia machine and patient breathing circuit.
- Examine the patient for signs of anaphylaxis (e.g., erythema, urticaria, hypotension, tachycardia).

Diagnostic Studies

- Auscultation of lung fields
- Chest X-ray
- Fiberoptic bronchoscopy

Subsequent Management

Treat the underlying problem. If difficulty persists, consider a pulmonary or critical care medicine consult.

Special Cases

Endobronchial Intubation

Inadvertent endobronchial intubation (usually into right mainstem bronchus) is common. Early detection and correction may decrease the risk of complications.

The average distance from the larynx to the carina is 12–14 cm and changes with age, height, and head position. The ETT moves cephalad when the neck is extended and caudad when the neck is flexed and can move as much as 5 cm with maximal cervical range of motion. Small changes in head position can cause endobronchial migration of the ETT in infants and small children. One-lung ventilation as a result of endobronchial ETT placement can cause atelectasis and hypoxemia.

Prevention

- Observe the endotracheal tube passing through the glottis and ensure that the upper end of the cuff is no more than 3–4 cm beyond the glottis.
- As a general rule, do not pass the endotracheal tube >21 cm in women and 23 cm in men (measured at the teeth) in average-sized patients.
- Ensure that equal, bilateral breath sounds are present.
- Chest X-ray should show the tip of the tube overlying the third or fourth thoracic vertebral body.
- Fiberoptic bronchoscopy should show the tip of the endotracheal tube 5–7 cm above the carina.

Bronchospasm

Bronchospasm is characterized by wheezing, increased airway pressures, and prolonged expiration time, and can be exacerbated by several triggers, including direct stimulation of the laryngeal and tracheal areas, histamine release associated with drug administration, and noxious stimuli. Bronchospasm should be recognized and treated promptly to avoid hypoxia, hypotension, and increased morbidity and mortality. Anaphylaxis as a cause of bronchospasm should also be included in the differential and ruled out.

Risk Factors: Bronchospasm

- Asthma
- Chronic obstructive pulmonary disease
- Anaphylaxis
- Smoking
- Light anesthesia
- Upper respiratory infection

Management

- Consider using a supraglottic airway instead of endotracheal intubation when appropriate.
- Use sevoflurane or isoflurane in at-risk patients.

- Increase the depth of anesthesia.
- Sevoflurane and isoflurane are potent bronchodilators.
- Ketamine and propofol have bronchodilating properties.
- Increase exhalation time to prevent auto-PEEP.
- Administer a β -agonist (e.g., albuterol MDI 8–10 puffs).
- Administer an inhaled anticholinergic (e.g., ipratropium 500 μ g by neb or MDI).
- Consider intravenous epinephrine (initial dose 5 to 10 μ g) in refractory or severe cases, especially if associated with anaphylaxis.
- Consider intravenous magnesium (2 g over 20 minutes) in severe cases.
- Consider administration of intravenous steroids (e.g., dexamethasone 10 mg). (Steroids take 4–6 hours to reach full effect.)
- If the patient with severe bronchoconstriction or status asthmaticus deteriorates, consider disconnecting the ventilator to allow adequate expiratory time.
- Ventilator settings may need to be adjusted with a long expiratory phase (1:3 or 1:4), low tidal volume (4–8 mL/kg), low rate (<14 bpm), no or low PEEP (<5) to prevent breath stacking and auto-PEEP, which will result in permissive hypercapnea.

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Hemoptysis

Definition

Massive hemoptysis is defined as >600 mL of blood loss in 24 hours, and exsanguinating hemoptysis is considered to be the loss of at least 1000 mL of blood at a rate of >150 mL/h.

Presentation

- Intubated patients: frothy or frank blood in the ETT.
- Chest X-ray may show signs of aspirated blood.

Etiology*Infection*

- Bronchitis
- Necrotizing pneumonia
- Lung abscess
- Tuberculosis
- Fungal infection
- Parasitic infection

Neoplastic

- Primary lung cancer
- Bronchial adenoma
- Metastatic lung cancer

22 **Cardiovascular**

- Pulmonary embolism
- Mitral stenosis
- Left ventricular failure
- Atrioventricular fistula
- Congenital heart disease
- Pulmonary hypertension

Pulmonary

- Cystic fibrosis
- Bronchiectasis
- Tuberculosis
- Trauma during intubation, endoscopy, or endobronchial surgery
- Related to tracheostomy
- Arteriovenous fistula
- Tumors

Hematologic

- Upper airway bleeding
- Disorders of coagulation
- Disseminated intravascular coagulopathy
- Thrombocytopenia
- Uremia
- Platelet dysfunction

Traumatic

- Aortic aneurysm
- Ruptured bronchus
- Chest injury
- Foreign body aspiration
- Tracheal-innominate artery fistula

Iatrogenic

- Bronchoscopy
- Lung biopsy
- Pulmonary artery catheterization
- Endobronchial brachytherapy
- Pulmonary hypertension

Alveolar Hemorrhage Syndromes

- Antiphospholipid syndrome
- Behçet syndrome
- Goodpasture syndrome
- Henoch-Schönlein purpura
- Systemic lupus erythematosus
- Wegener's granulomatosis
- Hematemesis and aspiration into the airway
- Idiopathic pulmonary hemosiderosis

Unknown Cause (Cryptogenic)

Immediate Management

- Increase FiO_2 to 100%.
- Ensure that adequate supplies of blood products are available. In the setting of exsanguinating hemoptysis, consider activating the massive transfusion protocol.
- Initiate aggressive resuscitation with intravenous (IV) fluids.
- Support blood pressure as needed with ephedrine (5 mg IV) or phenylephrine (100 mcg IV) boluses. If refractory, consider phenylephrine or epinephrine infusion.
- Control the airway—intubate if ETT is not in place.
- In most cases, a single lumen ETT facilitates evacuation of blood from the airway, bronchial lavage, and fiberoptic bronchoscopy.
- Convert to a double lumen endotracheal tube (DLT) or use a bronchial blocker (BB) to isolate the lungs if indicated (e.g., massive hemorrhage from below the carina).
- In an emergency when a DLT or a BB is not available, it is possible to push the ETT into a mainstem bronchus on the nonbleeding side to achieve lung isolation.

Immediate Management (continued)

- Rigid bronchoscopy identifies the site of bleeding and may allow immediate treatment by cauterization, ablation, or submucosal injection of vasoconstrictors.
- Consider extracorporeal membrane oxygenation (ECMO) as a bridge to definitive treatment in appropriate patients with life-threatening hemoptysis.

Diagnostic Studies

- Coagulation tests (PT/INR/aPTT/platelet count)
- Rigid/fiberoptic bronchoscopy
- Chest X-ray
- Computed tomography (CT) scan of the chest and neck as indicated
- Bronchial arteriogram
- Echocardiogram to rule out cardiac origin
- Right heart catheterization

Subsequent Management

- Correct any coagulation defects (see Coagulopathy, chapter 5).
- Recombinant activated factor VII is sometimes used with diffuse alveolar bleeding.
- Treat the primary cause of bleeding.
- Bronchial artery embolization can be effective in the management of life-threatening massive hemoptysis.
- A thoracic surgeon should be consulted if bronchial artery embolization is not feasible.
- Adopt a multidisciplinary approach to management of life-threatening massive intrapulmonary hemorrhage and hemoptysis.

Further Reading

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- Seijo LM, Daniel H, Sterman DH. Interventional pulmonology. *N Engl J Med.* 2001; 344(10): 740–749.
- Shigemura N, et al. Multidisciplinary management of life-threatening massive hemoptysis: a 10-year experience. *Ann Thorac Surg.* 2009; 87(3): 849–853.

High-Risk Tracheal Extubation**Definition**

Removal of the endotracheal tube from the airway in a patient who is at risk for respiratory complications.

Presentation

The risks of tracheal extubation are underestimated by many anesthesiologists. Although this is a routine procedure in most patients, extubation can cause serious morbidity and mortality. Closed claims data suggest that 12% of claims related to difficult airways occurred at extubation and 5% at recovery. Although the final decision to extubate is made at the end of the surgical procedure or in the ICU or postanesthesia care unit (PACU), planning should begin in the preoperative period and continues through the post-extubation phase.

- Patients can be classified as “low risk” or “high risk.”
- Tracheal extubation is an elective procedure.
- Published guidelines can be used as a framework to guide decision making.

Pathophysiology and Relative Contraindications

- Removal of the endotracheal tube decreases the anesthesiologists’s ability to control the airway.
- Surgery and anesthesia can negatively impact airway patency.
- Residual anesthesia, opioids, and neuromuscular blockade place the patient at risk for hypoventilation.
- Blunted airway reflexes impair the patient’s ability to manage his or her airway.
- Reduced functional residual capacity, V/Q mismatch, hypoventilation, diffusion hypoxia, and atelectasis may cause hypercarbia or hypoxemia.
- Airway injury or edema
- Cardiovascular instability
- Neurologic dysfunction
- Metabolic derangement
- Electrolyte disturbance
- Lack of availability of adequate equipment and/or skilled personnel
- Patient position
- Reduced access to the airway (e.g., dressings, gastric tubes, cervical collar)
- Interruption of oxygen supply during transfer

Immediate Management

- Determine whether the patient is at low or high risk for airway complications after extubation.
- Confirm safety of tube removal. A key question is: Is it safe to remove this tube?

Immediate Management (continued)

- Increase FiO_2 to 100%.
- Suction the airway.
- Position the patient appropriately.
- Rule out residual neuromuscular blockade.
- Establish regular spontaneous breathing and adequate ventilation.
- Use a bite block. If the patient occludes the tube, consider deflating ETT or SGA cuff to prevent negative pressure edema.
- The patient should open his or her eyes and follow commands.
- Minimize head and neck movements.
- Apply positive pressure, deflate the cuff, and remove the ETT while lung volumes are near vital capacity.
- Deliver 100% oxygen and confirm airway patency with an anesthetic breathing system.
- Continue delivering oxygen by face mask until recovery is complete.
- Consider placement of an airway exchange catheter (AEC) before extubating high-risk patients.
- Consider placement of SGA as a bridge to extubation.

Diagnostic Studies

- Examine the airway for edema, bleeding, blood clots, trauma, foreign bodies, or anatomic changes.
- Consider direct or indirect laryngoscopy.
- Perform cuff-leak test.
- Consider chest X-ray.
- Consider gastric decompression by OG/NG tube if gastric distention is present.
- Monitor neuromuscular blockade (via twitch monitor or accelerometer).

Airway Exchange Catheters

- Airway should be suctioned and lidocaine can be administered through ETT.
- When the patient meets criteria for extubation, lubricated airway exchange catheter (AEC) is placed through the tracheal tube to predetermined depth.
- Placement of the AEC should be to a maximum depth of 25 cm (to avoid injury to the lung).
- Remove the tracheal tube over the AEC while maintaining AEC position.
- Secure AEC to the cheek or forehead with tape.
- Clearly label the AEC to prevent confusion with the OG/NG tube.

- Monitor patient in the PACU or ICU while the AEC is in place.
- Supplemental oxygen can still be given by face mask, nasal cannula, or continuous positive airway pressure (CPAP) as needed.
- The patient should remain NPO until the AEC is removed.
- If the AEC triggers coughing, ensure that the tip is above the carina and administer lidocaine via AEC.
- Remove the AEC when the airway is no longer at risk.
- If reintubation is necessary, the patient can be intubated over the AEC.

Risk Factors: High-Risk Extubation

- Known difficult airway
- Airway deterioration (bleeding, edema, trauma)
- Restricted airway access
- Obesity/OSA
- Aspiration risk
- Uncertain ability to oxygenate
- Potentially difficult reintubation
- General risk (cardiovascular, respiratory, neurological, metabolic, special surgical, or medical)

Prevention

Recognition of extubation as a high-risk phase of anesthesia and appropriate perioperative planning are critical to prevent airway-related morbidity and mortality. Identification of low- and high-risk extubations and continuous reassessment are critical to this process.

Further Reading

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Intrinsic Upper Airway Obstruction

Definition

Partial or complete airway blockage that increases upper airway resistance (above the larynx) and may cause impaired oxygenation and ventilation.

Presentation

- Dyspnea
- Hypercarbia or hypoxemia (may cause obtundation)
- Snoring
- Wheezing
- Stridor
- Use of accessory muscles of respiration
- Tracheal tug (downward movement of trachea with inspiration)
- Retractions at sternal notch
- Apnea
- Agitation
- Thoraco-abdominal dyssynchrony

Pathophysiology

Airway obstruction in the spontaneously breathing patient has many causes, including aspirated foreign bodies, infections (e.g., epiglottitis [Figure 2.1], diphtheria, Ludwig's angina), laryngospasm, bronchospasm, tumor or a hematoma impinging on the airway, airway trauma, tonsillar hypertrophy, obstructive sleep apnea, nasopharyngeal and oral packing, and airway edema (e.g., anaphylaxis, smoke inhalation, burn injury).

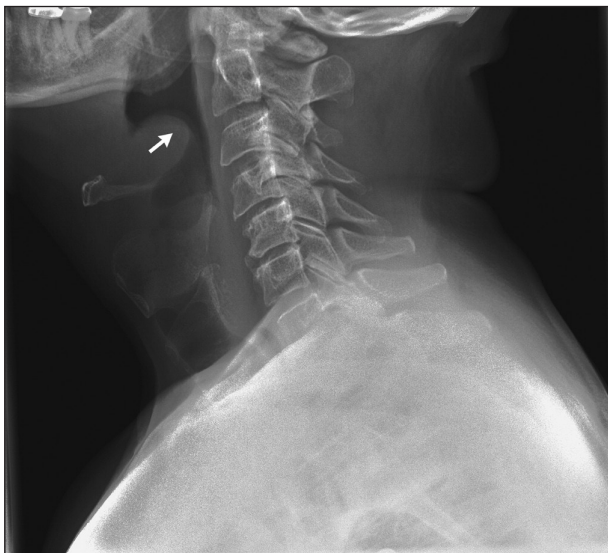


Figure 2.1 Thumb sign epiglottitis.

DIFFERENTIAL DIAGNOSIS

- Snoring/obstructive sleep apnea
- Bronchospasm
- Bradypnea or apnea from drug overdose or other causes
- Laryngospasm

Immediate Management

- Increase FiO_2 to 100%.
- Attempt to open the airway with a jaw thrust, a nasopharyngeal airway, an oropharyngeal airway, or a supraglottic airway device.
- If possible, administer continuous positive airway pressure (CPAP).
- Consider nebulized racemic epinephrine and/or intravenous dexamethasone (10 mg IV in an adult).
- Consider lightening or reversing sedation.
- Consider nasoendoscopy to assist in diagnosis of etiology.
- Consider endotracheal intubation for unremitting obstruction.
- Be prepared for surgical airway as primary or secondary intervention.
- Consider a helium oxygen mixture as a bridge therapy in stridorous patients if intubation is not feasible.

Further Reading

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Laryngospasm

Definition

Closure of the upper airway caused by glottic muscle spasm.

Presentation

- Difficult or impossible face mask ventilation
- Difficult or impossible ventilation with a supraglottic airway
- “Crowing” sound on inspiration

Pathophysiology

Laryngospasm is especially common in children and is associated with light planes of anesthesia and irritation of the vocal cords by foreign matter (e.g., blood or secretions).

DIFFERENTIAL DIAGNOSIS

- Bronchospasm
- Stridor
- Foreign body in the airway
- Airway obstruction from edema, infection, tumor, hematoma, etc.

Immediate Management

- Administer 100% oxygen with positive pressure ventilation.
- If caused by light anesthesia, administer propofol or other drugs to deepen the level of anesthesia.
- Consider succinylcholine 0.1 mg/kg IV.

Special Considerations

- Untreated laryngospasm can rapidly lead to hypoxemia and hypercarbia.
- Patients who generate high negative inspiratory pressures while attempting to breathe against the obstruction may develop negative-pressure pulmonary edema.

Further Reading

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Ludwig's Angina

Definition

Ludwig's angina is a multispace infection of the floor of the mouth. The infection usually starts with infected mandibular molars and spreads to the sublingual, submental, buccal, and submandibular spaces.

Presentation

- Signs of airway obstruction, such as the use of the accessory muscles of respiration
- Dyspnea

- Drooling
- Edema and distortion of airway structures, especially when associated with fever and leukocytosis

Pathophysiology

The tongue becomes elevated and displaced posteriorly, which may lead to obstructive apnea, especially when the patient is in the supine position.

DIFFERENTIAL DIAGNOSIS

- Retropharyngeal abscess
- Submandibular abscess
- Epiglottitis
- Dental abscess

Immediate Management

- Airway management depends on clinical severity, surgical preferences, and other factors (e.g., CT scan or MRI findings).
- If the clinical situation permits, transport the patient to the operating room for airway management.
- **A skilled surgeon and emergency cricothyroidotomy equipment must be present before intubation is attempted.**
- Consider performing an awake fiberoptic intubation if at all possible (see page 396). Elective tracheostomy prior to incision and drainage may be necessary in the setting of significant airway compromise.

31

Subsequent Management

- Initiate antibiotic therapy, either empiric or based on culture and sensitivity testing.
- Transfer the patient to the ICU until edema resolves and extubation can be safely achieved.

Special Considerations

- Loss of the airway is the leading cause of death in patients with Ludwig's angina.
- Extubation may be hazardous. Special precautions, such as the use of a tube exchange catheter, may be appropriate.
- The abscess may rupture spontaneously or after attempts at laryngoscopy and intubation, flooding the hypopharynx with pus that may then be aspirated.

Further Reading

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Rapid-Sequence Intubation

Definition

A technique of inducing general anesthesia so as to reduce the risk of pulmonary aspiration of gastric contents. An induction agent (e.g., propofol or etomidate) is administered, immediately followed by a short-acting neuromuscular blocking agent (e.g., succinylcholine or rocuronium) to quickly render the patient unconscious and chemically paralyzed to block the active vomiting reflex and facilitate airway management. Pressure is usually applied at the cricoid cartilage (Sellick's maneuver) to compress the esophagus, reducing the risk of passive regurgitation.

Indications

- Emergency surgery in a patient who has not been fasting
- Patients with paralytic ileus or acute abdomen
- Patients with significant reflux or achalasia cardia
- Patients with acute trauma requiring immediate surgery
- Women presenting for surgery in the last trimester of pregnancy

Contraindications

- Patients with anticipated or known difficult airway
 - Situations in which laryngeal injury may be present
- Consider awake intubation in these situations.

The Rapid Sequence Induction (“The 9 Ps”)

Preparation

Prepare all necessary equipment and drugs, and have a backup plan.

- A working laryngoscope and different types of blades.
A video laryngoscope may be desirable if a difficult airway is anticipated.
- ETTs of the desired size with smaller ETTs available. A stylet should be inserted prior to inducing anesthesia.
- Device to confirm proper placement of ETT (e.g., capnograph)
- Working suction
- Gum elastic introducer (Bougie) or Eschmann stylet
- Appropriately sized SGA for rescue

- Equipment for emergency tracheotomy/cricothyrotomy where appropriate
- Functional IV access
- Appropriate monitoring equipment

Patient Evaluation

- Evaluate the airway to rule out possible difficult ventilation or intubation.
- Review possible contraindications to medications.

Preoxygenation

Administer 100% oxygen for 3–5 minutes with a tight seal around the mask. If the patient is cooperative, five vital-capacity breaths are nearly as effective.

Premedication

This should be used judiciously because it may increase the risk of aspiration or delay awakening in the event that the patient cannot be intubated.

- Midazolam: 0.02–0.05 mg/kg. Use with caution in patients with head injury or those who may need to be awakened rapidly.
- Fentanyl: 3 mcg/kg IV 2–3 minutes prior
- Lidocaine: 1.5 mg/kg IV 2–3 minutes prior
- Consider aspiration prophylaxis such as sodium citrate.

Paralysis and Induction

Rapidly administer an anesthetic followed by a neuromuscular blocking agent. Do not titrate medication to effect.

- Choose an induction agent:
 - Etomidate: 0.3 mg/kg IV
 - Ketamine: 1–2 mg/kg IV
 - Propofol: 1–2 mg/kg IV
- Choose a neuromuscular blocking agent:
 - Succinylcholine: 1–2 mg/kg
 - Rocuronium: 1–1.2 mg/kg

Position and Protect the Patient

- Position the head and neck into the sniffing position by flexing the neck and extending the atlanto-occipital joint. Reposition the head if an adequate view of the glottic opening is not achieved.
- If the patient is in a cervical spine collar, an assistant must maintain inline stabilization and the front of the collar must be removed.
- Apply cricoid pressure (Sellick's maneuver) before induction. Do not release the cricoid pressure until correct ETT position is confirmed.

- Wait for 45–60 seconds to allow full effect of the neuromuscular blockade.
- If the patient will tolerate apnea, do not ventilate him or her at this time to prevent gaseous distention of the stomach.

Pass the Endotracheal Tube

Visualize the tube going through the vocal cords.

Proof of Placement

Establish that the ETT is in the correct position by end-tidal capnography, bilateral breath sounds, chest rise, and fogging within the ETT.

Postintubation Care

- Ventilate.
- Secure the ETT.
- Evacuate the stomach.
- Administer postintubation sedation if out of the operating room (OR).
- Maintain appropriate postintubation hemodynamics.

Further Reading

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Chapter 3

Respiratory Emergencies

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Acute Lung Injury and Acute Respiratory Distress Syndrome	36
Bronchospasm	38
Decreased ETCO_2 (Intraoperative)	40
Difficult Controlled Ventilation	42
Hemoptysis	44
Hypercarbia (Intraoperative)	46
Hypoxemia (Intraoperative)	48
Pneumothorax	50
Pulmonary Edema	52
Pulmonary Thromboembolism	54
Respiratory Precautions	57

Acute Lung Injury and Acute Respiratory Distress Syndrome

Definition

Acute onset of bilateral pulmonary infiltrates on noted chest X-ray with pulmonary edema, poor systemic oxygenation, and absence of left atrial hypertension.

Presentation

Patients with acute lung injury (ALI) or acute respiratory distress syndrome (ARDS) are often intubated and mechanically ventilated. Their physical exam is notable for reduced breath sounds and possibly wheezing. Increased peak inspiratory and plateau pressures may be seen with positive pressure ventilation. Arterial blood gases may show hypercarbia in the setting of poor lung compliance.

Pathophysiology

The early phase of ARDS is characterized by pulmonary capillary leak and interstitial and alveolar edema. There is a loss of surfactant activity. During the late phase of ARDS, pulmonary fibrosis and decreased lung compliance can develop.

Immediate Management

- Increase FIO_2 and titrate positive end-expiratory pressure (PEEP) to maintain adequate oxygenation.
- Consider ventilation with low tidal volumes (4–8 cc/kg predicted body weight).
- Avoid plateau pressures >30 cm H_2O .
- Permissive hypercapnia may be necessary.
- Consider alternative ventilatory strategies (e.g., airway pressure release ventilation [APRV]).

DIFFERENTIAL DIAGNOSIS

- Pulmonary edema
- Multilobar pneumonia
- Diffuse alveolar hemorrhage
- Pneumonitis
- Pulmonary embolus
- Transfusion related acute lung injury (TRALI)
- Bronchiolitis obliterans-organizing pneumonia (BOOP)

Diagnostic Studies

- Chest X-ray (shows patchy infiltrates that extend to the periphery)

- Right heart catheterization (CVP or pulmonary artery catheter)
- Thoracic ultrasound shows B-lines (suggests an interstitial process)

Subsequent Management

- Treat the precipitating cause of ALI/ARDS.
- Employ mechanical ventilation as necessary to manage respiratory failure.
- Transfer the patient to the intensive care unit (ICU) for further management.
- Neuromuscular blockade may rarely be required to facilitate ventilation and oxygenation.
- Consider the prone position and nitric oxide therapy for refractory hypoxemia.
- Consider extracorporeal membrane support if medical management and mechanical ventilation fail.

Risk Factors

- Sepsis
- Pneumonia
- Pneumonitis
- Pancreatitis
- Toxic drug reaction
- Inhalational injury
- Massive transfusion
- Mechanical ventilation

37

Prevention

Early and aggressive treatment of precipitating causes may prevent progression to lung injury.

Special Considerations

- Patients who come to the operating room with ARDS can present with increased peak airway pressures and high levels of positive end-expiratory pressure (PEEP).
- Patients often require specialized transport from an ICU (see Transportation of a Critically Ill Patient).
- The anesthesiologist must be familiar with the patient's mode of ventilation in order to ensure safe transport. Arterial blood gas samples guide changes to ventilator strategy in the operating room.

Further Reading

Brodie D, Bacchetta M. Extracorporeal membrane oxygenation for ARDS in adults. *N Engl J Med*. 2011; 365: 1905–1914.

Jason D, Christie P, Lanken, N. Acute lung injury and the acute respiratory distress syndrome. In: Hall JB, Schmidt G, Wood LDH, eds. *Principles of Critical Care*. 3rd ed. New York: McGraw-Hill; 2005:515–547.

Slutsky AS, Ranieri VM. Ventilator-induced lung injury. *N Engl J Med*. 2013; 369: 2126–2136.

Bronchospasm

Definition

Spasmodic contraction of bronchial smooth muscle.

Presentation

Decreased SpO_2 or an upsloping of the ETCO_2 tracing on the capnograph. An increase in peak inspiratory pressure (PIP) may also be seen if the patient is mechanically ventilated. Visible slowing or lack of chest fall may be observed. Wheezing or decreased breath sounds may be heard. Hypotension is a late sign in severe bronchospasm due to hypoxia or auto-PEEP, which decreases venous return.

38 Pathophysiology

Bronchospasm can occur after a mechanical (intubation) or chemical (anaphylatoxin) stimulus activates mast cells, eosinophils, lymphocytes, epithelial cells, and macrophages to release various mediators, such as histamine, to constrict bronchial smooth muscle. The hyper-irritable airway is often edematous and produces mucus, which further increases airway resistance and can lead to mucus plugging.

DIFFERENTIAL DIAGNOSIS

- Mechanical obstruction (e.g., kinked endotracheal tube, mucus plug)
- Pulmonary edema
- Tension pneumothorax
- Aspiration pneumonia
- Pulmonary embolus
- Pulmonary edema
- Endobronchial intubation

Immediate Management

- Increase FIO_2 to 100%.
- Increase the inspired concentration of a potent volatile anesthetic if one is being used (bronchodilator properties).
- Administer β -agonist bronchodilators (e.g., nebulized albuterol).
- Consider epinephrine 10–30 mcg IV in refractory cases, titrated to effect.

Diagnostic Studies

Clinical presentation. No specific diagnostic studies.

Subsequent Management

- If surgery has not started, consider postponing an elective procedure in the setting of unremitting severe bronchospasm.
- Consider administration of steroids (e.g., hydrocortisone 100 mg IV).
- Maintain an adequate depth of anesthesia to prevent further bronchospasm.
- Avoid unnecessary airway manipulation.
- Avoid triggering agents, such as histamine-releasing drugs.
- Consider postoperative mechanical ventilation.

Risk Factors

- History of asthma, chronic obstructive pulmonary disease (COPD), emphysema
- Recent upper airway infection
- Airway manipulation
- Can occur in healthy patients

Prevention

- If the clinical situation permits, avoid endotracheal intubation in at-risk patients.
- Consider use of a regional anesthesia technique if the patient has a history of reactive airway disease.
- Patients with a history of asthma have bronchial hyper-reactivity, and may benefit from preoperative corticosteroid treatment.
- Intravenous agents, including propofol, ketamine, and lidocaine, may decrease airway resistance.

Special Considerations

- Even with adequate preparation and implementation of preventive measures, bronchospasm may still occur in the operating room. Avoid elective surgery within 10–14 days of an upper respiratory infection, as the airways can be hyper-reactive during this time period.

Further Reading

Woods BD, Sladen RN. Perioperative considerations for the patient with asthma and bronchospasm. *Br J Anaesthesiol.* 2009; 103(S1): i57–i65.

Decreased ETCO_2 (Intraoperative)

Definition

End tidal CO_2 <30 mm Hg

Presentation

A decrease in ETCO_2 in a mechanically ventilated patient may occur with either hypocarbia (hyperventilation) or hypercarbia (inability to eliminate CO_2). A sudden decrease in ETCO_2 may indicate cardiovascular collapse or an embolic phenomenon.

Etiology

Most commonly caused by hyperventilation in a mechanically ventilated patient. It may also reflect increased dead space with a normal PaCO_2 . Sudden, catastrophic decrease in cardiac output will decrease the ETCO_2 because of decreased perfusion (CO_2 is not being carried to lungs).

Immediate Management

- Assess cause of decreased ETCO_2
- Send arterial blood to determine PaCO_2
- Sudden:
 - Consider cardiovascular collapse.
 - Assess perfusion by other means (blood pressure, presence of a pulse oximeter waveform).
 - Assess volume status.
 - Consider providing cardiovascular support using inotropes or vasopressors.
 - If an embolic event is suspected:
 - Adjust head of bed down.
 - Flush the surgical field with saline if air embolus is suspected.
 - Provide hemodynamic support, e.g., epinephrine 4 mcg/min titrate as necessary.
- Gradual:
 - Decrease minute ventilation (if patient is being ventilated).

DIFFERENTIAL DIAGNOSIS

- Air leak in sample line
- Gas analyzer error
- Low ETCO_2 with hypocarbia ($\text{PaCO}_2 <35$ mm Hg)
- Hyperventilation
 - High minute ventilation in a mechanically ventilated patient
 - Pain

- Anxiety
- Compensation for metabolic acidosis
- Low ETCO_2 with hypercarbia ($\text{PaCO}_2 > 45$ mm Hg)
 - Pulmonary thromboembolus
 - Air embolus
 - Fat embolus
 - CO_2 embolus (laparoscopic surgery)
 - Amniotic fluid embolus
- Obstruction
 - Mechanical (kinking of tube)
 - Bronchospasm
 - COPD
- Low cardiac output state
- Esophageal intubation

Diagnostic Studies

- Arterial blood gas (ABG) to determine PaCO_2
- Transthoracic or transesophageal echocardiogram to assess cardiac function. Note: An echocardiogram will also detect bubbles if a venous air embolus is the cause.
- Spiral computed tomography (CT) if a thromboembolic event is suspected

Subsequent Management

- Correct the underlying cause.
- Provide hemodynamic support.
- Intubate the trachea and initiate mechanical ventilation.

Risk Factors

- Sitting craniotomy or any surgical procedure in which the operative site is above the heart: air embolus
- Bone cement implantation: fat embolus (see Bone Cement Implantation Syndrome).
- Hemorrhagic or cardiogenic shock: low cardiac output state

Further Reading

Eisenkraft J, Leibowitz A. Hypoxia and equipment failure. In: Yao F, ed. *Anesthesiology: Problem-Oriented Patient Management*. 5th ed. New York: Lippincott Williams & Wilkins; 2003:1116–1136.

Difficult Controlled Ventilation

Definition

Inability to effectively oxygenate and/or ventilate a patient who is mechanically ventilated.

Presentation

High peak airway pressures and hypercarbia are observed with difficult controlled ventilation. Patients are often hypoxemic may be hypotensive because increased intrathoracic pressure can decrease venous return.

Etiology

- High peak airway pressures can be caused by poor compliance of the lung parenchyma and increased resistance to airflow due to:
 - Mucous plug
 - Bronchospasm
 - Pulmonary edema
 - Auto-PEEP
 - Pneumothorax
- Elevated plateau pressure (the pressure applied to the small airways and the alveoli during positive pressure) suggests poor lung compliance.
- Low peak airway pressures and loss of measured tidal volumes may indicate an air leak in the circuit or in the tracheobronchial tree (i.e., a bronchopulmonary fistula).

Immediate Management

- Increase FiO_2 to 100% and titrate PEEP to maintain adequate oxygenation.
- Auscultate breath sounds.
- Begin manual ventilation.
- Suction the endotracheal tube.
- Administer a bronchodilator if bronchospasm is suspected (albuterol 2–4 puffs into the ETT).
- Administer a diuretic if pulmonary edema is present (furosemide 20 mg IV).
- Exclude anesthesia gas machine or ventilator failure.
- Increase the level of sedation and consider neuromuscular blockade if necessary.

DIFFERENTIAL DIAGNOSIS

- Increased resistance to flow
- Bronchospasm
- Obstruction
 - Kinked endotracheal tube
 - Mucus plug
 - Decreased lung compliance
 - Inadequate muscle relaxation
 - Tension pneumothorax
 - Auto-PEEP
 - Acute lung injury/acute respiratory distress syndrome
 - Pulmonary edema
 - Aspiration
 - Opioid-induced chest wall rigidity
 - Pulmonary hemorrhage
 - Intra-abdominal hypertension/abdominal compartment syndrome
 - High insufflation pressures in laparoscopic surgery

Diagnostic Studies

- Measure peak airway pressures.
- Measure plateau pressure.
- Perform chest X-ray.
- Perform thoracic ultrasonography.

Subsequent Management

- Decrease tidal volume to limit volutrauma.
- Increase respiratory rate to ensure adequate minute ventilation.
- Monitor for evidence of auto-PEEP (sudden hypotension, clinical evidence of “breath stacking”).
- Consider CT scan of the chest if the underlying cause is unknown.
- Treat the underlying cause of decreased compliance.
- If conventional mechanical ventilation is inadequate, consider high frequency oscillatory ventilation (HFOV), airway pressure release ventilation (APRV), or extracorporeal membrane oxygenation (ECMO).

Risk Factors

- Patients with COPD are at risk for auto-PEEP.
- Patients with diffuse pulmonary injury (infection, sepsis) may develop ARDS as their disease progresses.

Prevention

- Maintain adequate sedation.
- Monitor for signs of auto-PEEP.

Special Considerations

- Patients who are difficult to ventilate may have increased peak airway pressure, require high levels of PEEP, and often require transport to or from an ICU. These patients may require deep sedation and an ICU ventilator in order for safe transport from the ICU to the Operating Room. The anesthesiologist must be familiar with the patient's mode of ventilation in order to ensure a safe transport (see Transporting the Critically Ill Patient). Arterial blood gas samples can guide changes in ventilator strategy in the operating room.

Further Reading

Brodie D, Bacchetta M. Extracorporeal membrane oxygenation for ARDS in adults. *N Engl J Med*. 2011; 365: 1905–1914.

Schmidt G. Ventilator waveforms: clinical interpretation. In: Hall JB, Schmidt G, Wood LDH, eds. *Principles of Critical Care*. 3rd ed. New York: McGraw-Hill; 2005:427–443.

Hemoptysis

Definition

Cough productive of blood or bloody sputum. *Massive hemoptysis* is the production of 300–600 cc of blood in a 12- to 24-hour period.

Presentation

Reduced breath sounds, frank blood in the sputum, diffuse pulmonary infiltrates on chest X-ray. In an intubated patient, blood may appear in the endotracheal tube. Hypoxia and increased peak inspiratory pressures may occur during positive pressure ventilation in patients with massive hemoptysis. Coagulopathy can trigger hemoptysis, and anemia may be present. Hemoptysis may be the presenting symptom for pulmonary infection or malignancy.

Pathophysiology

Disruption of the pulmonary vessels lining the trachea, bronchi, or alveoli caused by infection, tumor, vascular disorders, or trauma. Coagulopathy may exacerbate bleeding.

DIFFERENTIAL DIAGNOSIS

- Nasal trauma
- Pharyngeal trauma
- Gastrointestinal bleeding

Immediate Management

- Increase FiO_2 and titrate PEEP to maintain oxygenation.
- Inspect the nose and pharynx to exclude an upper-airway source of bleeding.
- Consider the possibility of a gastrointestinal source of bleeding.
- Consider bronchoscopy to identify the bleeding site.
- Consider inserting a double lumen endotracheal tube or bronchial blocker to isolate the bleeding site (see page 407).
- If hemoptysis is life-threatening, consider endobronchial ablation, bronchial artery embolization, external beam irradiation, or surgical resection.

Diagnostic Studies

Chest CT and bronchoscopy may localize the source of pulmonary bleeding.

Subsequent Management

- Identify and treat the cause of a coagulopathy.
- Consider a surgical consultation if bronchial embolization is unsuccessful or multiple bleeding vessels are seen with angiography.
- Consider a course of steroids in patients with vasculitis.
- Consider plasmapheresis in patients with Goodpasture syndrome.

Risk Factors

- Infection
- Tumor
- Pulmonary vascular abnormalities
- Cardiac causes (e.g., mitral stenosis)
- Trauma
- Coagulopathy
- Pulmonary–renal syndromes (Goodpasture syndrome)

Prevention

- Avoid unnecessary instrumentation of the airway.
- Correct underlying coagulopathy (see Coagulopathy).

Special Considerations

- Frequent suctioning of the endotracheal tube may cause hemoptysis.

Further Reading

Albert R. Massive hemoptysis. In: Hall JB, Schmidt G, Wood LDH, eds. *Principles of Critical Care*. 3rd ed. New York: McGraw-Hill; 2005:583–585.

Sakr L, Dutau H. Massive hemoptysis: an update on the role of bronchoscopy in diagnosis and management. *Respiration*. 2010; 80(1): 38–58.

Hypercarbia (Intraoperative)

Definition

Increased arterial partial pressure of carbon dioxide ($\text{PaCO}_2 > 45 \text{ mm Hg}$).

Presentation

Tachycardia, agitation, hypertension, and eventually obtundation.

Etiology

Hypercarbia is caused by either hypoventilation or increased CO_2 production. Hypoventilation due to decreased respiratory drive or airway obstruction in sedated patients often leads to hypercarbia. Poor lung compliance may reduce minute ventilation and cause hypercarbia. Residual anesthetic effects or inadequate reversal of muscle relaxants can cause postoperative hypercarbia. Splinting due to pain can lead to increased dead space, hypoventilation, and hypercarbia. Hypermetabolic states and fever may contribute to increased CO_2 production.

Immediate Management

- Intubate the trachea and initiate mechanical ventilation for severe respiratory acidosis, if the patient is unable to protect his or her airway, or if respiratory failure is imminent.
- Consider noninvasive positive pressure ventilation (CPAP, BiPap) if airway protection is not required.
- Increase minute ventilation to reduce PaCO_2 .
- Ask the surgeon to lower insufflation pressure during laparoscopic surgery.
- In a spontaneously breathing patient, consider judicious reversal of opioids with naloxone (naloxone 0.04-mg IV increments).

DIFFERENTIAL DIAGNOSIS

- Hypoventilation
 - Low minute ventilation
 - Narcotics or oversedation
 - Inadequate reversal of muscle relaxant
 - Splinting
- Malignant hyperthermia (see Special Considerations).
- Bronchospasm (COPD or asthma exacerbation)
- Acute lung injury, acute respiratory distress syndrome
- Severe pneumonia
- Aspiration
- Shivering
- Sepsis
- CO₂ insufflation during laparoscopy
- Bicarbonate administration
- Thyrotoxicosis

Diagnostic Studies

Arterial blood gas (ABG) analysis to quantify degree of hypercarbia and acidosis.

Subsequent Management

- Treat the underlying cause of hypercarbia.

Risk Factors

- Laparoscopic surgery (insufflation of peritoneal cavity with CO₂)
- Obesity
- Obstructive sleep apnea (OSA)
- Chronic CO₂ retainers
- COPD
- Asthma
- Poor lung compliance
- Narcotic administration

Prevention

- Judicious use of narcotics and other sedatives
- Adequate reversal of muscle relaxants
- Adequate minute ventilation, especially in laparoscopic surgery

Special Considerations

- Hypercarbia causes respiratory acidosis that cannot be compensated for in the acute period. Hypercarbia may cause severe hypertension, hyperkalemia, arrhythmias, myocardial

depression, altered mental status, increased intracranial pressure, and increased pulmonary vascular resistance.

- Rapidly rising ETCO_2 in conjunction with tachycardia and rising temperature may be caused by malignant hyperthermia (MH). Malignant hyperthermia must be diagnosed quickly and treatment initiated immediately (see Malignant Hyperthermia).

Further Reading

Lane J. Postoperative respiratory insufficiency. In: Atlee JL, ed. *Complications in Anesthesia*. 2nd ed. Philadelphia: Saunders Elsevier; 2007:877–880.

Hypoxemia (Intraoperative)

Definition

Decreased partial pressure of oxygen in the blood ($\text{PaO}_2 < 60$ mm Hg) often manifested by a decrease in SpO_2 .

Presentation

Decreased SpO_2 , cyanosis, and possibly hypertension and agitation. Left untreated, hypoxemia may progress to hypotension, bradycardia, arrhythmias, and neurologic and myocardial ischemia.

Etiology

- Oversedation and/or narcotic overdose can cause hypoventilation and airway obstruction in patients undergoing surgery with monitored anesthetic care (MAC).
- Decrease in functional residual capacity (FRC)
- Position (supine position decreases FRC)
- Atelectasis and alveolar shunting
- Blunted hypoxic pulmonary vasoconstriction (may be caused by inhaled anesthetics)
- Ventilation perfusion mismatching
- Intrapulmonary shunt caused by mainstem bronchus intubation
- Anesthesia machine malfunction

Immediate Management

- Increase FIO_2 to 100% and titrate PEEP while assessing patient.
- Auscultate the lung fields to assess breath sounds.
- Check ETCO_2 to ensure that the ETT is in the trachea and that the lungs are being ventilated.
- If the patient is not already intubated, consider intubation and mechanical ventilation for severe hypoxia or if respiratory failure is imminent.

DIFFERENTIAL DIAGNOSIS

- Esophageal intubation
- Mechanical disconnect from ventilator or O₂ source
- Right mainstem intubation
- Airway obstruction
- Hypoventilation
- Atelectasis
- Presence of a mucus plug
- Bronchospasm
- Pneumothorax
- Pulmonary embolus
- Pulmonary edema
- Acute lung injury
- Aspiration
- Low cardiac output state

Diagnostic Studies

- Arterial blood gas analysis (ABG) to quantify PaO₂
- Chest X-ray
- Consider bronchoscopy (assess ETT placement, find a possible obstruction).
- Thoracic ultrasonography

Subsequent Management

- Increase FiO₂ to maintain oxygenation.
- Titrate PEEP to improve oxygenation.
- Treat the underlying cause of hypoxemia.
- Prepare for endotracheal intubation and mechanical ventilation.
- Consider nitric oxide therapy for refractory hypoxemia.
- Consider extracorporeal membrane oxygenation for refractory hypoxemia.

Risk Factors

- Underlying pulmonary disease
- Obstructive sleep apnea
- Aspiration risk
- Use of narcotics
- Advanced age
- Obesity
- Shivering

Prevention

- Confirm ETT position with capnography and auscultation.
- Use narcotics and sedatives carefully in patients who are breathing spontaneously.
- Ensure that the patient is adequately ventilated.

Special Considerations

- Intraoperative hypoxemia is one of the most common problems that an anesthesiologist encounters, and should be considered life threatening. Prompt diagnosis and treatment are essential to preventing further complications, such as hypotension, arrhythmias, and end-organ damage.

Further Reading

Lane J. Postoperative respiratory insufficiency. In: Atlee JL, ed. *Complications in Anesthesia*. 2nd ed. Philadelphia: Saunders Elsevier; 2007:877–880.

Pneumothorax

Definition

Presence of gas, usually air, in the pleural cavity that leads to collapse of the lung. This condition may be life threatening if the gas cannot escape (i.e., tension pneumothorax).

Presentation

A small pneumothorax is often asymptomatic. As the pneumothorax becomes larger, hypoxia, tachypnea, tachycardia, and chest pain may occur. It may be possible to hear hyper-resonance on the affected side with percussion. Decreased or absent breath sounds may also be heard on the affected side. Increased peak airway pressures and plateau pressures occur in mechanically ventilated patients. A tension pneumothorax is often associated with hypotension.

Pathophysiology

A tension pneumothorax occurs when a one-way valve mechanism occurs after injury to the pleural space. With each inspiration, gas is trapped in the pleural space causing collapse of the lung. If intrapleural pressure increases significantly, mediastinal shift causes kinking of major veins at the thoracic inlet of the neck and inferior vena cava, resulting in decreased venous return and hypotension.

Immediate Management

- Increase FIO_2 to 100%.
- Decompress the pleural space by inserting a large bore needle in the mid-clavicular line in the second intercostal space (Figure 3.1).
- Insert a chest tube.

DIFFERENTIAL DIAGNOSIS

- Hemothorax
- Mucus plug
- Endobronchial intubation
- Severe bronchospasm

Diagnostic Studies

- Auscultation (absent breath sounds on the affected side)
- Percussion (hyper-resonance).
- Chest X-ray (lung collapse and mediastinal shift)
- Thoracic ultrasonography (presence of lung point; absence of lung sliding)

Subsequent Management

- Bronchoscopy if bronchial tree injury is suspected
- Surveillance chest X-ray to evaluate progression of pneumothorax
- Chest tube management

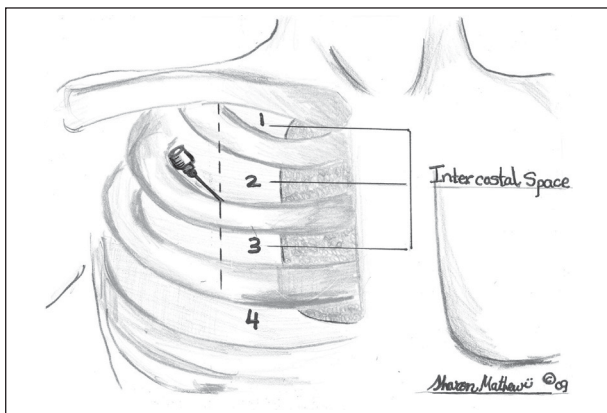


Figure 3.1 Decompression of tension pneumothorax.

Risk Factors

- Central line insertion
- Laparoscopic surgery
- Excessive tidal volume or peak airway pressure
- Obstructed chest tube (e.g., kink or clot)
- Pulmonary blebs

Prevention

- Avoid excessive tidal volume or peak airway pressure.
- Chest X-ray or imaging after central line placement to prevent progression of a small pneumothorax to a tension pneumothorax.

Special Considerations

- Tension pneumothorax should be considered in patients who develop a pulseless electrical activity (PEA) cardiac arrest.

Further Reading

Ali J. Torso trauma. In: Hall JB, Schmidt G, Wood LDH, eds. *Principles of Critical Care*. 3rd ed. New York: McGraw-Hill; 2005:1421–1441.

Pulmonary Edema**Definition**

The abnormal accumulation of extravascular fluid in the lung parenchyma.

Presentation

The first signs of pulmonary edema in an anesthetized patient are often hypoxemia and decreased SpO₂. Rales or wheezing are heard over the lung fields. Frothy sputum may be noted in the endotracheal tube. In an awake patient, respiratory distress, tachycardia, and agitation. Jugular venous distention may be seen on physical examination.

Pathophysiology

- High pulmonary and venous hydrostatic pressure (cardiogenic) or increased capillary permeability (noncardiogenic).
- Cardiogenic pulmonary edema is caused by impaired venous drainage from the pulmonary vasculature to the left atrium. This often occurs when the left atrial pressure is high in the setting of left ventricular dysfunction and/or valvular abnormalities.

- Negative pressure or postobstruction pulmonary edema occurs when negative intrapleural pressure increases the pulmonary hydrostatic pressure gradient, causing fluid to move from the pulmonary vasculature to the interstitium.

Immediate Management

- Increase FIO_2 to 100% and titrate PEEP to maintain oxygenation.
- Initiate diuresis (start with furosemide 20 mg IV).
- Intubate the trachea and begin positive pressure ventilation if the patient is hypoxic or respiratory failure is imminent.
- If cardiogenic pulmonary edema is suspected, consider afterload reduction with nitroglycerine (infusion starting at 0.5 mcg/kg/min) and support blood pressure with vasopressors (see Congestive Heart Failure).
- Treat the underlying cause.

DIFFERENTIAL DIAGNOSIS

- Aspiration pneumonitis
- Acute lung injury/ARDS
- Neurogenic pulmonary edema
- Aspiration pneumonitis
- Fat embolism
- TACO (transfusion-associated circulatory overload)
- TRALI (transfusion-related acute lung injury)

Diagnostic Studies

- Chest X-ray (bilateral pulmonary infiltrates and edema around pulmonary arteries)
- Pulmonary artery catheter or echocardiogram can differentiate between cardiogenic and noncardiogenic pulmonary edema.
- Thoracic ultrasonography (presence of B lines)

Subsequent Management

- Ventilatory support, including PEEP
- Serial arterial blood gas measurements
- Central venous pressure monitoring may aid in medical management, for example, diuresis.

Risk Factors

- Cardiogenic
 - Systolic dysfunction
 - Diastolic dysfunction

Risk Factors (continued)

- Volume overload
- Myocardial infarction
- Valvular abnormalities
- Respiratory
 - Negative pressure
 - Laryngospasm
 - Upper airway obstruction
 - Upper airway tumor or foreign body
 - Tonsillar hypertrophy

Prevention

- Avoid fluid overload in a patient with compromised myocardial function.
- Ensure adequate perfusion pressure and avoid tachycardia in patients with coronary artery disease.
- Identify patients at risk for airway obstruction.

Special Considerations

- Negative pressure pulmonary edema often resolves within 24 hours. Cardiogenic pulmonary edema may occur 2–3 days postoperatively when fluids are mobilized.

Further Reading

Ali J. Special considerations in the surgical patient. In: Hall JB, Schmidt G, Wood LDH, eds. *Principles of Critical Care*. 3rd ed. New York: McGraw-Hill; 2005:1321–1330.

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Pulmonary Thromboembolism**Definition**

Obstruction of a pulmonary artery or one of its branches, most commonly by a venous thrombus that becomes dislodged and eventually travels to the lungs. Pulmonary embolus may also be caused by fat, air, carbon dioxide, or amniotic fluid emboli.

Presentation

The signs and symptoms may be subtle. Small emboli may go undetected; unexplained fever, tachycardia, and rales may be the only presenting symptoms. Some patients may present with a triad of dyspnea, hemoptysis, and chest pain. Patients with large emboli may present with sudden hypoxia, hypercarbia, tachypnea, decreased ETCO_2 , and circulatory collapse.

Pathophysiology

Pulmonary thromboembolism occurs when a venous thrombus is dislodged and travels to the lungs. This occurs most commonly in the setting of venous stasis or injury, and most thrombi originate in the lower extremity deep vein system. Pulmonary embolism can also be caused by air, fat, carbon dioxide, or amniotic fluid emboli. Gas exchange becomes impaired as dead space increases. A widened alveolar to arterial gradient is often seen. Right ventricular afterload increases.

DIFFERENTIAL DIAGNOSIS

- Thromboembolism
- Air embolism
- Fat embolism
- Amniotic fluid embolism
- Acute myocardial infarction
- Severe bronchospasm
- Anaphylaxis
- Pneumothorax

55

Immediate Management

- Increase FIO_2 to 100% to maintain oxygenation.
- Consider intubation and mechanical ventilation if hypoxia is severe.
- Support the circulation with fluid, vasopressors, and inotropes.
- Consider right ventricular afterload reduction with nitric oxide.
- Begin systemic anticoagulation if not contraindicated and a thromboembolism is diagnosed (heparin 80 U/kg IV bolus, then 18 U/kg per h IV, titrated to therapeutic INR)
- NOTE: Consider thrombolytic therapy or embolectomy if the patient is hemodynamically unstable.

Diagnostic Studies

- Helical chest computed tomography angiography
- D-dimer is of limited value, as levels are often elevated in the perioperative setting.

- Ventilation-perfusion scan
- Noninvasive venous Doppler studies to assess for deep venous thrombosis
- Echocardiography to evaluate for right ventricular dilation or strain

Subsequent Management

- Identify the source of the embolism.
- Continue anticoagulation if indicated.
- Consider inserting an inferior vena cava filter if anticoagulation is contraindicated.

Risk Factors

- Thromboembolism
 - Malignancy
 - Surgery and trauma
 - Immobility
 - Pregnancy
 - Hypercoagulable states
 - Obesity
 - Indwelling central lines
- Other embolic events
 - Sitting craniotomy (air embolus)
 - Laparoscopic surgery (CO₂ embolus)
 - Hip replacement (fat embolus)

Prevention

- Intermittent compression stockings
- Subcutaneous (SC) heparin (e.g., 5000 U SC) in high-risk patients
- Early mobilization after surgery

Special Considerations

- The decision to administer anticoagulants or thrombolytics may be complicated in postoperative patients, and must take into consideration the risk of postoperative bleeding.
- Brain natriuretic peptide levels predict right ventricular dysfunction and mortality.
- Pulmonary embolus should be considered in patients with PEA.

Further Reading

Jaff MR, McMurtry MS, Archer SL, et al. Management of massive and sub-massive pulmonary embolism, iliofemoral deep vein thrombosis, and

Respiratory Precautions

Bioterrorism, severe acute respiratory syndrome (SARS), multi-drug resistant tuberculosis, and H1N1 influenza have brought new concerns to the health care provider, and especially the anesthesiologist. Anesthesiologists must provide rapid and appropriate care when managing an airway, while at the same time making sure to protect themselves from communicable diseases.

The Centers for Disease Control and Prevention (CDC) recommends use of N95 respirator masks while caring for patients with suspected H1N1. The N95 mask is tighter-fitting than a traditional face mask (Figure 3.2). An N95 respirator covers the nose and mouth and is designed to have a tight fit. If worn correctly, it should filter out at least 95% of particles as small as $0.3\ \mu\text{m}$.

The use of eye protection in the form of fluid shields or goggles is also recommended to protect against contact with sputum, gastric contents, or other bodily fluids while securing a patient's airway. Additionally, wearing a gown may further protect the anesthesiologist as well as other patients.

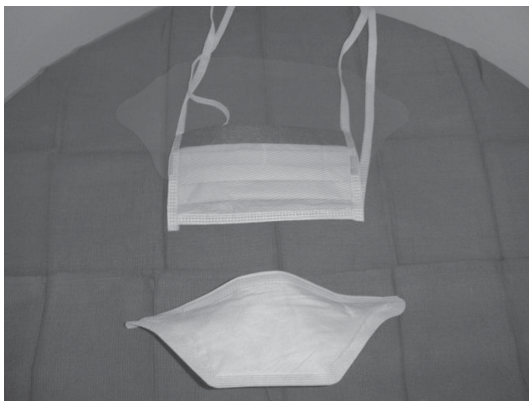


Figure 3.2 Standard fluid shield mask (top) and N95 respirator (bottom).

If a bioterrorism strike is suspected, gown, gloves, N95 respirators, and fluid shields should be worn. Immediately contact local authorities and the CDC for likely pathogens and take further proper precautions, including potentially a splash protective suit and a self-contained breathing apparatus.

Chapter 4

Cardiovascular Emergencies

Ajoy Katari and Benjamin Kohl

Arrhythmias: Asystole	60
Arrhythmias: Atrial Fibrillation	61
Pharmacologic Rate Control (Goal <110 bpm)	62
Arrhythmias: Bradycardia	64
Arrhythmias: Narrow Complex Tachycardia	66
Arrhythmias: Ventricular Fibrillation	68
Arrhythmias: Wide Complex Tachycardia	69
Cardiac Tamponade	72
Cardiac Trauma	74
Congestive Heart Failure	75
Hypertension	77
Hypotension	79
Myocardial Ischemia	81
Postoperative Hemorrhage	84
Pulmonary Embolism	85
Thoracic Aortic Dissection	87
Valvular Disease: Aortic Regurgitation	89
Valvular Disease: Aortic Stenosis (AS)	91
Valvular Disease: Mitral Regurgitation (MR)	93
Valvular Disease: Mitral Stenosis (MS)	94
Venous Gas Embolism	96

Arrhythmias: Asystole

Definition

Complete absence of electrical and mechanical cardiac activity.

Presentation

Usually preceded by other arrhythmias. Excessive vagal stimulation can sometimes be the initial trigger, for example, insufflation of the abdominal cavity for laparoscopic surgery or excessive intraocular pressure.

Pathophysiology

Primary asystole has a poor prognosis. Bradycardia is usually caused by conditions such as hypoxia, hyperkalemia or hypokalemia, acidosis, or myocardial infarction.

Immediate Management

- Confirm asystole in >1 electrocardiogram lead.
- Begin cardiopulmonary resuscitation (CPR).
- Establish an airway (endotracheal intubation preferred).
- Administer epinephrine (1 mg every 3–5 minutes) OR
- Administer vasopressin (40 U within the first 10 minutes of CPR).
- Administer atropine (1 mg every 3–5 minutes for three doses showed earlier recovery to circulation, but no significant difference in 30-day survival or neurologic outcome).

DIFFERENTIAL DIAGNOSIS

- Monitor lead disconnection
- Severe bradycardia
- Ventricular fibrillation
- Low voltage electrocardiogram (ECG)

Diagnostic Studies

- ECG
- Echocardiogram

Subsequent Management

- Employ reverse of underlying causes (e.g., hypoxia, acidosis).
- Administer sodium bicarbonate to correct acidosis.
- Consider temporary transcutaneous or transvenous cardiac pacing.

Risk Factors

- Hypoxia
- Electrolyte imbalances (e.g., hyperkalemia)

- Hypovolemia
- Hypothermia
- Ocular surgery
- Hypersensitive carotid sinus
- Underlying cardiac conduction abnormalities

Prevention

Early identification of underlying conditions such as hypoxia, acidosis, and ensuing arrhythmias.

Special Considerations

- Transcutaneous pacing has not been shown to favorably effect survival unless instituted early and is generally considered to be ineffective therapy.
- Several factors need to be considered to terminate resuscitation efforts. The most commonly used parameters include
 - Duration of resuscitative effort >30 minutes without sustained return of spontaneous circulation (ROSC)
 - Asystole as initial rhythm
 - Advanced age with severe comorbid disease
- Ensure normothermia before terminating resuscitation.

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 7.2: Management of cardiac arrest. *Circulation*. 2005; 112: IV-58–IV-66. Atropine sulphate for patients with out-of-hospital cardiac arrest due to asystole and pulseless electrical activity. *Circ J*. 2011; 75(3): 580.

Arrhythmias: Atrial Fibrillation

Definition

An irregularly irregular heart rhythm with the absence of P waves on ECG. Most common cardiac arrhythmia.

Presentation

- May be asymptomatic
- Palpitations
- Chest pain/angina
- Congestive heart failure
- Syncope
- Transient ischemic attacks

Pathophysiology

Atrial fibrillation may often occur in patients with otherwise normal hearts, but is also associated with excessive alcohol consumption, hypertension, hyperthyroidism, and myocardial ischemia.

Atrial dilatation and fibrosis are the changes that occur most commonly in patients with atrial fibrillation.

Immediate Management

- Administer synchronized DC cardioversion 150 J (may increase to 360 J) if the patient is unstable, including hypotension, chest pain, lightheadedness.
- Consider DC cardioversion in stable patients if <48 hours after onset.

Pharmacologic Rate Control (Goal <110 bpm)

- Beta-blockers (e.g., metoprolol 2.5 mg intravenously [IV] to initiate therapy)
- Calcium channel blockers (diltiazem 0.25 mg/kg IV \times 1 dose)
- Amiodarone (IV dose of 150 mg \times 1 over 10 minutes, follow 1 mg/min for 6 hours, and 0.5 mg/min for 18 hours)
- Digoxin (only preferred when in heart failure or as second-line therapy)

DIFFERENTIAL DIAGNOSIS

- Sinus tachycardia
- Multifocal atrial tachycardia
- Reentrant tachycardia
- Junctional tachycardia
- Atrial flutter

Diagnostic Studies

- ECG
- Electrophysiology study
- Echocardiogram
- Holter monitoring

Subsequent Management

- Refer for electrophysiology study
- If atrial fibrillation (AF) continues, request a cardiology consultation for antiarrhythmic therapy or DC cardioversion

- Electrophysiology and cryoablation
- Surgical ablation
- If AF continues for >48 hours, initiate anticoagulation to decrease the risk of stroke

Risk Factors

- Age (8% of people >80)
- Recent cardiothoracic surgery (particularly valvular surgery)
- Atrial dilatation
- Hyperthyroidism
- Males are at greater risk than females
- Smoking
- Alcohol consumption
- Coronary artery disease
- Stress

Prevention

- Avoid discontinuing beta-blockers in the perioperative period
- Maintain normal electrolyte values (particularly potassium and magnesium)

Special Considerations

- Atrial fibrillation increases the risk of stroke by 700%.
- Cardioversion is indicated for atrial fibrillation (<48 hours after onset) or if a transesophageal echocardiogram shows no evidence of thrombus.
- Atrioventricular (AV) nodal blockers (Verapamil, Digoxin) are contraindicated in atrial fibrillation with pre-excitation rhythms (e.g., AV nodal re-entry or Wolff-Parkinson-White syndrome). Check for delta-wave on ECG.

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 7.3: Management of symptomatic bradycardia and tachycardia. *Circulation*. 2005; 112: IV-67–IV-77.

Echahidi N. Mechanisms, prevention, and treatment of atrial fibrillation after cardiac surgery. *J Am Coll Cardiol*. 2008; 51: 793–801.

National Institute for Health and Clinical Excellence Guidelines for Atrial Fibrillation. <http://www.nice.org.uk/nicemedia/pdf/CG036niceguideline.pdf> Accessed August 9, 2009.

Arrhythmias: Bradycardia

Definition

A heart rate <60 beats per minute.

Presentation

- Hypotension
- Nausea
- Altered mental status
- Pulmonary edema
- Chest pain

Pathophysiology

May be secondary to cardiac ischemia, atrioventricular node disease, hypoxemia, acidosis, or drugs (e.g., narcotics, beta-blockers).

A sick sinus node or conduction abnormalities may cause bradycardia. The preceding presentations are suggestive of organ malperfusion.

Immediate Management

- If an iatrogenic cause is suspected (e.g., pressure on the globe, traction on a hernia sac), inform the surgeon and ask him or her to stop.
- Administer atropine (0.5 mg IV, repeat every 5 minutes to a total dose of 3 mg).
- If bradycardia is unremitting, begin transcutaneous pacing if there is evidence of tissue hypoperfusion (see page 411).

DIFFERENTIAL DIAGNOSIS

- Excessive beta or calcium channel blocker therapy
- Excessive vagal stimulation
- High dose narcotic administration
- Hypersensitivity of carotid sinus
- Excessive intraocular pressure
- Elevated systemic vascular resistance
- Hypoxia
- Acidosis
- Cardiac tamponade
- Electrolyte abnormalities

Diagnostic Studies

- ECG
- Echocardiogram
- Angiogram (if acute myocardial infarction is suspected).

Subsequent Management

- Begin transvenous pacing if transcutaneous pacing fails to capture (see page 416).
- Administer epinephrine (2 to 10 mcg/min) or Dopamine (2–10 mcg/kg/min) infusion.
- Refer the patient for permanent pacemaker implantation.

Risk Factors

- Obstructive sleep apnea
- Pharmacologic (alpha-agonists, beta-blockers, calcium channel blockers, narcotics)
- Ocular surgery
- Surgery near carotid sinus (e.g., carotid endarterectomy)
- Laparoscopic surgery
- Age
- Elevated blood pressure
- Elevated cholesterol
- Smoking
- Heavy alcohol consumption
- Use of recreational drugs
- Psychological stress or anxiety

Prevention

- Maintain normal electrolytes.
- Ask the surgeon to inject the carotid sinus with lidocaine during carotid endarterectomy.
- Use alpha-agonist agents cautiously.

Special Considerations

- Atropine works at the AV node and is unlikely to be effective on bradycardia due to a block below the bundle of His. Glucagon 3 mg IV followed by a 3-mg/h drip can be used if bradycardia is attributed to beta-blocker therapy.

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular Care. Part 7.3: Management of symptomatic bradycardia and tachycardia. *Circulation*. 2005; 112: IV-67–IV-77.

Arrhythmias: Narrow Complex Tachycardia

Definition

Heart rate >100 beats per minute (bpm) (may be regular or irregular) with a narrow QRS complex (<120 msec)

Presentation

- Hypotension
- Palpitations
- Altered mental status
- Chest pain
- Pulmonary edema

Pathophysiology

A narrow QRS complex implies rapid activation of the ventricles via the normal His-Purkinje system and suggests that the arrhythmia originates within or above the AV node (i.e., supraventricular). Narrow QRS complex tachycardia is most commonly either sinus or reentrant tachycardia (e.g., Wolff-Parkinson-White syndrome).

Immediate Management

- Administer adenosine (6 mg rapid IV push, repeat 12 mg \times 2).
- Stable regular narrow complex tachycardia: Treat underlying cause (e.g., fever, anemia, shock, sepsis, pain).
- Stable irregular narrow-complex tachycardias: Control heart rate with diltiazem (15 mg IV over 20 minutes) or metoprolol (5 mg IV).
- Consider DC cardioversion if hemodynamically unstable.
- Unstable non-sinus arrhythmias must be treated with DC cardioversion.
- Unstable sinus arrhythmias can be treated with beta-blockers. (Consider a short-acting agent such as esmolol 1000 mcg/kg IV bolus for immediate or 500 mcg/kg bolus for more gradual control.)

DIFFERENTIAL DIAGNOSIS

- Sinus tachycardia
- Atrial tachycardia
- Multifocal atrial tachycardia
- Re-entrant tachycardia (e.g., Wolff-Parkinson-White syndrome)
- Junctional tachycardia

- Atrial fibrillation
- Atrial flutter

Diagnostic Studies

- ECG
- Echocardiography

Subsequent Management

- If adenosine fails, initiate rate control with either intravenous calcium-channel blockers or betablockers (diltiazem 0.25 mg/kg IV \times 1, escalate to 0.35 mg/kg IV after 15 minutes if unsuccessful on first dose).
- Chemical cardioversion: Administer procainamide (50 mg/min IV, up to a dose of 18–20 mg/kg) or amiodarone (5 mg/kg IV over 15 minutes).
- Perform DC cardioversion for tachycardia resistant to pharmacologic interventions and/or in patients who are hemodynamically unstable.
- Refer to the cardiology service for electrophysiology testing and possible ablation.

Risk Factors

- Fever
- Inadequate anesthesia
- Hypovolemia
- Myocardial ischemia
- Hyperthyroid
- Vagolytic drugs

Prevention

- Maintain normothermia
- Maintain normovolemia (adequate fluid resuscitation)
- Avoid discontinuing beta-blockers in the perioperative period
- Avoid using vagolytic drugs (e.g., atropine, pancuronium)

Special Considerations

- Regular narrow complex tachycardia is most often sinus tachycardia.
- Irregular narrow complex tachycardia may be atrial flutter, atrial fibrillation, or multifocal atrial tachycardia.
- Transient chest discomfort, dyspnea, and flushing may occur when adenosine is administered.
- Consider the risk of embolic stroke before cardioversion.

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 7.3: Management of symptomatic bradycardia and tachycardia. *Circulation* 2005; 112: IV-67–IV-77.

Arrhythmias: Ventricular Fibrillation

Definition

A nonperfusing rhythm of the ventricles with disorganized and non-coordinated electrical activity.

Presentation

Either a witnessed cardiac arrest or noted on the ECG.

Pathophysiology

Usually secondary to myocardial ischemia or underlying conditions such as hypoxia and acidosis.

Immediate Management

- Deliver a 360 J monophasic (200 J biphasic) cardioversion: may repeat every 2 minutes if necessary. (**Avoid delay—administer shocks before airway management.**)
- If the airway is not secured, ventilate the patient with a self-inflating bag and mask. Intubate the trachea and ventilate with a self-inflating bag when feasible.
- Begin cardiopulmonary resuscitation after shock to maintain 30:2 ratio for 2 minutes **without pausing to check rhythm or pulse.**
- Administer epinephrine 1 mg IV (repeat every 3–5 minutes) OR 40 U IV vasopressin (single dose, replaces first or second dose of epinephrine).

DIFFERENTIAL DIAGNOSIS

- Drug toxicity (cocaine, digitalis, antidepressants)
- Pulmonary embolism
- Hypoxia
- Acidosis
- Cardiac tamponade
- Electrolyte abnormalities (hyperkalemia/hypokalemia)
- Hypovolemia
- Shivering

Diagnostic Studies

- ECG
- Serum electrolyte levels

- Arterial blood gas
- Cardiac enzymes
- Echocardiogram

Subsequent Management

- Airway management, secure endotracheal tube, and place on ventilator
- ACLS secondary survey
- Administer amiodarone 300 mg IV. (Repeat 150 mg in 3–5 minutes if VF/PVT persists.)
- Administer lidocaine (if amiodarone unavailable) 1.0–1.5 mg/kg IV, may repeat to a 3 mg/kg max loading dose.
- Administer magnesium sulfate 1–2 g IV diluted in 10 mL D₅W for torsades de pointes or suspected/known hypomagnesemia.

Risk Factors

- Cardiac comorbidities
- Advanced disease states

Prevention

- Early detection of precipitating underlying conditions and therapy.

Special Considerations

- ACLS guidelines have been updated to emphasize the importance of early defibrillation, hence the recommendation to deliver 360 J instead of an escalating energy. Current guidelines recommend cardioversion **before** definitive airway management.

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 7.2: Management of cardiac arrest. *Circulation*. 2005; 112: IV-58–IV-66.

Arrhythmias: Wide Complex Tachycardia

Definition

Heart rate >100 beats per minute (may be regular or irregular) with a wide QRS complex (>120 msec).

Presentation

- Hypotension
- Altered mental status
- Palpitations

- Chest pain
- Pulmonary edema

Pathophysiology

A widened QRS complex implies slow ventricular activation. This arrhythmia commonly originates outside of the normal conduction system but may be ventricular or supraventricular. A regular, wide-complex tachycardia is most often ventricular tachycardia.

Immediate Management

- Synchronized DC cardioversion if the patient is hemodynamically unstable (start at 100 J and escalate to 200 J, then 300 J, and 360 J)

Regular Rhythm

- Administer amiodarone (150 mg IV given over 10 minutes, repeated as needed to a total of 2.2 g IV over the first 24 hours)

Irregular Rhythm

- Administer procainamide (20 mg/min IV up to a total of 17 mg/kg)
- Administer lidocaine (1 mg/kg IV, may repeat 0.5 mg/kg every 5 minutes as needed to total of 3 mg/kg)

DIFFERENTIAL DIAGNOSIS

Regular

- Ventricular tachycardia
- supraventricular tachycardia with aberrant conduction
- Monitoring artifact (e.g., while scrubbing a surgical site)
- Paced rhythm with atrial tachycardia (atrial sense, ventricular paced)

Irregular

- Atrial fibrillation with aberrancy (bundle branch block),
- Atrial fibrillation with pre-excitation (e.g., Wolff-Parkinson-White syndrome)
- Polymorphic ventricular tachycardia

Diagnostic Studies

- ECG
- Echocardiogram
- Electrophysiology testing

Subsequent Management

- Perform DC cardioversion (start at 100 J and increase to 360 J) for regular tachycardia that is resistant to pharmacologic intervention or if the patient is hemodynamically unstable.
- In a history of pre-excitation (e.g., Wolff-Parkinson-White syndrome), or evidence of pre-excitation on the ECG (e.g., delta wave), administer procainamide (20 mg/min continuous infusion until the arrhythmia is suppressed, the patient is hypotensive, the QRS widens 50% beyond baseline, or a maximum dose of 17 mg/kg is administered).

Risk Factors

- Age >50 years
- Myocardial ischemia or old myocardial infarction
- Known re-entrant pathway

Prevention

- Maintain normal electrolyte levels (particularly potassium and magnesium)
- Anti-arrhythmic (Class I Na⁺ channel for recurrent cases, Class II beta blockers reduce mortality from Myocardial Infarction, and class III K⁺ channel may be beneficial for diabetics) drugs
- Refer for catheter ablation
- Refer for implantable cardioverter defibrillator (ICD) implantation
- Change pacemaker to VVI (or AAI) in at-risk patients

Special Considerations

- AV nodal blockers are contraindicated in wide complex, irregular tachycardia, especially when the etiology is unknown because they may precipitate ventricular fibrillation. If the patient is stable, further diagnosis is warranted as AVNRT and AVRT (both are types of SVT) require different management and can present as wide complex tachycardia. Although amiodarone is used as an alternative treatment in wide complex tachycardia, it may cause irregular wide complex dysrhythmias due to pre-excitation. Converting to unstable ventricular tachycardia (VT) or ventricular fibrillation (VF).

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 7.3: Management of symptomatic bradycardia and tachycardia. *Circulation* 2005; 112:IV-67–IV-77.

Cardiac Tamponade

Definition

Compression of the heart due to accumulation of fluid in the pericardial space. (The normal volume of fluid in the pericardial sac is 25–50 mL.) Gradual accumulation allows time for compensation and may not result in symptomatic tamponade; rapid accumulation may be life threatening.

Presentation

- Dyspnea
- Orthopnea
- Tachycardia
- Jugular venous distention
- Distant heart sounds
- Pulsus paradoxus (systolic blood pressure decreases by at least 10 mm Hg during inspiration)
- Beck's triad (small, quiet heart, rising venous pressure, and hypotension)

Pathophysiology

External pressure on the heart reduces the ventricular preload.

Immediate Management

- In the setting of profound hypotension, emergency pericardiocentesis may be necessary.
- Consider IV fluid administration to increase preload.
- Maintain heart rate to maintain cardiac output.
- Maintain normal sinus rhythm.

DIFFERENTIAL DIAGNOSIS

Note: Some of these conditions can cause tamponade.

- Acute myocardial infarction
- Postoperative bleeding
- Aortic dissection
- Iatrogenic (e.g., catheter insertion)
- Connective tissue disorders
- Uremia
- Positive end expiratory pressure (auto-PEEP)

Diagnostic Studies

- Electrical alternans on ECG (variation of R-wave axis with alternate beats)

- Globular heart on chest radiography
- Accumulation of fluid seen on echocardiography
- Equalization of diastolic pressures in all four chambers of the heart

Subsequent Management

- Consult a cardiac surgeon for pericardiectomy.
- Consider using ketamine for induction of general anesthesia (may help to maintain heart rate and blood pressure).
- Maintain spontaneous ventilation until the pericardial sac is opened.
- If mechanical ventilation is necessary, use low tidal volume combined with a high rate to minimize intrathoracic pressure.
- Keep the heart rate high.
- Maintain adequate preload.

Risk Factors

- Trauma
- Myocardial infarction
- Connective tissue disorders
- Uremia

Prevention

- Treat uremic patients early.
- Ensure early recognition and control of cardiac surgical bleeding.
- Maintain a high index of suspicion for cardiac injury during pacemaker electrode implantation or removal.
- Early recognition and intervention are critical to successful management.

Special Considerations

- Pneumothorax and cardiac perforation may occur during a cardiac window procedure.
- Pulmonary edema and global systolic dysfunction may occur after drainage of pericardial fluid.

Further Reading

Grocott HP, Gulati H, Srinathan S, Mackensen GB. Anesthesia and the patient with pericardial disease. *Can J Anaesth*. 2011; 58(10): 952–966.

Soler-Soler J, et al. Management of pericardial effusion. *Heart*. 2001; 86: 235–240.

Cardiac Trauma

Definition

Penetrating or blunt injury to the myocardium.

Presentation

- Dyspnea
- Tachycardia
- Chest pain
- Flail chest

Pathophysiology

Severe blunt injury, usually caused by a high-impact force, that causes injury to thoracic organs.

Immediate Management

- Assess airway, breathing, and circulation.
- Provide supplemental O₂ as required to maintain oxygenation.
- Intubate the trachea and initiate mechanical ventilation if the patient is in respiratory distress. Note: Airway management may be complicated by concomitant injury to the trachea. Rule out tracheal injury if intubating the patient with direct laryngoscopy.
- Request a surgical consultation for chest tube insertion and/or pericardiocentesis.
- Emergency surgery, possibly requiring cardiopulmonary bypass, may be necessary.
- Provide supportive management as required.

DIFFERENTIAL DIAGNOSIS

- Pulmonary or myocardial contusion
- Pneumothorax
- Esophageal tear/rupture
- Myocardial laceration
- Cardiac tamponade (see page 72)
- Thoracic aorta dissection
- Coronary artery laceration
- Diaphragmatic injury

Diagnostic Studies

- Chest X-ray and CT scan
- Bronchoscopy

- Esophagoscopy
- Transesophageal echocardiogram (TEE)

Subsequent Management

- Request a surgical consultation for possible chest exploration.
- Evaluate the tracheobronchial tree to rule out a concomitant lung injury.
- Use lung isolation techniques as required to optimize ventilation.

Risk Factor

- Trauma

Prevention

Although cardiac trauma is not truly a preventable phenomenon, early recognition and diagnosis of underlying pathology followed by timely intervention may reduce morbidity and mortality.

Special Considerations

- The use of nitrous oxide should be avoided because an as-yet undiagnosed pneumothorax may exist. Be alert for impaired ventilation of the dependent lung during one-lung ventilation; it may be caused by a tension pneumothorax. Some degree of cardiac injury should be anticipated in patients with blunt chest injury. Monitor the ECG continuously in the postoperative period.

Further Reading

- Bastos R, et al. Penetrating thoracic trauma. *Semin Thorac Cardiovasc Surg.* 2008; 20: 19–25.
- Singh KE, Baum VC. The anesthetic management of cardiovascular trauma. *Curr Opin Anaesthesiol.* 2011; 24(1): 98–103.
- Sybrandy KC, et al. Diagnosing cardiac contusion: old wisdom and new insights. *Heart.* 2003; 89: 485–489.

Congestive Heart Failure

Definition

A structural or a functional cardiac disorder with impaired ability of the ventricle to fill with (diastolic) or eject (systolic) blood.

New York Heart Association (NYHA) classification of severity:

- Class I—symptoms of heart failure (HF) only at activity levels that would limit normal individuals

- Class II—symptoms of HF with ordinary exertion
- Class III—symptoms of HF with less than ordinary exertion
- Class IV—symptoms of HF at rest

Presentation

- Dyspnea
- Fatigue
- Edema

Pathophysiology

Systolic heart failure is idiopathic in approximately 50% of patients. Etiologies include myocarditis, ischemic heart disease, infiltrative disease (amyloidosis), peripartum cardiomyopathy, hypertension, human immunodeficiency virus (HIV) infection, connective tissue disease, substance abuse, and drugs (e.g., doxorubicin). Diastolic heart failure include all of these, and hypertrophic and restrictive cardiomyopathies.

Immediate Management

- Increase FiO_2 to maintain oxygenation.
- Administer a loop diuretic (e.g., furosemide 20–40 mg IV).
- Consider an ACE inhibitor (enalapril 2.5 mg IV every 6 hours).
- Consider nitroglycerine (infusion starting at 0.5 mcg/kg/min, increase every 3–5 minutes to desired effect).
- Consider nesiritide (2 mcg/kg IV bolus, then 0.01 mcg/kg/min to a maximum of 0.03 mcg/kg/min).
- Consider beta-blockers (esmolol 500 mcg/kg over 1 minute, then 50 mcg/kg/min to a maximum of 300 mcg/kg/min).
- If the patient is awake and can take oral medication, consider angiotensin II receptor blockers (candesartan).
- Consider an aldosterone antagonist (i.e., spironolactone).

DIFFERENTIAL DIAGNOSIS

- Myocardial ischemia
- Primary pulmonary pathology
- Cardiomyopathy

Diagnostic Studies

- Chest X-ray with cardiomegaly, Kerley B lines, pleural effusions
- ECG to rule out ischemic or hypertrophic changes
- Echocardiogram to evaluate cardiac function
- Serum brain natriuretic peptide (BNP) level (elevated in heart failure)

Subsequent Management

- Correction of contributing systemic disease
- Lifestyle modification
- Discontinue drugs implicated in HF
- Specialized management for HF that is refractory to pharmacologic therapy:
 - Implantable cardioverter-defibrillator
 - Cardiac resynchronization therapy
 - Intra-aortic balloon pump (for life-threatening congestive heart failure [CHF])
 - Implantation of left ventricular assist device
 - Referral for heart transplantation

Risk Factors

- Coronary artery disease
- Smoking
- Hypertension
- Obesity
- Valvular heart disease

Prevention

Careful fluid management in patient at risk for CHF.

Special Considerations

- Implantable cardiac defibrillators detect and treat arrhythmias associated with heart failure, and may also be used for cardiac resynchronization therapy with biventricular pacing.
- Cardiac wraps have been used to stop further dilation of the ventricles, preventing deterioration of heart function.

Further Reading

Hunt SA, et al. 2009 Focused update incorporated into the ACC/AHA 2005 guidelines for the diagnosis and management of heart failure in adults: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines developed in collaboration with the International Society for Heart and Lung Transplantation. *J Am Coll Cardiol.* 2009; 53(15): e1–e90.

Hypertension

Definition

A blood pressure $>160/100$ in a patient who is otherwise normotensive.

Presentation

- Chest pain
- Headache
- Palpitations
- Stroke
- Pulmonary edema

Pathophysiology

The pathophysiology of hypertension is often multifactorial. Anything that increases preload, afterload, or contractility can cause hypertension.

Immediate Management

- Increase depth of anesthesia.
- Check for medication error.
- Administer incremental doses of a beta-blocker (e.g., labetalol 5 mg IV) if the heart rate is >60 . **Note:** Labetalol is preferred because it has both alpha- and beta-blocking properties.
- If the heart rate is <60 , administer hydralazine 5–10 mg IV in incremental doses.
- Administer nicardipine: Start at 5 mg/h IV and increase by 2.5 mg/h every 5–15 minutes. Maximum dose is 15 mg/h.
- Administer sodium nitroprusside for acute, life-threatening hypertension: Start at 0.3 mcg/kg/min and increase slowly. The maximum dose is 10 mcg/kg/min.

DIFFERENTIAL DIAGNOSIS

- Inadequate anesthetic depth
- Agitation
- Vasopressor error (inadvertent administration or wrong dose)
- Pheochromocytoma
- Thyrotoxicosis
- Aortic cross-clamp
- Elevated intracranial pressure
- Transection of the spinal cord at or above T5
- Eclampsia in pregnancy
- Electroconvulsive therapy

Diagnostic Studies

- ECG
- Echocardiogram
- Intra-arterial catheter for continuous blood pressure monitoring

Subsequent Management

- Request an internal medicine or cardiology consultation for workup and chronic treatment with long-acting beta-blockers, ACE inhibitors, angiotensin receptor blockers.
- Administer anxiolytics if appropriate.

Risk Factors

- Age >60 years
- Males are at greater risk than are females.
- Race
- Weight
- Stress
- Alcohol
- Smoking
- Family history
- Renal disease
- Diabetes
- Sleep apnea

Prevention

- Maintain adequate depth of anesthesia.
- Avoid rapid termination of antihypertensive medications.

Special Considerations

- An acute rise is of more significance than the actual numbers, but systolic values >220 mm Hg and diastolic values >120 mm Hg must be treated promptly.
- Elevated blood pressure in patients with chronic hypertension should be decreased by no more than 20%–30% of baseline to compensate for a rightward shift of the autoregulation curve.

Further Reading

Laslett L. Hypertension-preoperative assessment and perioperative management. *West J Med.* 1995; 162: 215–219.

Varon J. The diagnosis and treatment of hypertensive crisis. *Postgrad Med.* 2009; 121: 5–13.

Hypotension

Definition

Low systemic blood pressure that may be insufficient to maintain end-organ perfusion. Criteria vary with the patient and surgical setting, but commonly accepted definitions include:

- Systolic blood pressure <80 mm Hg
- Mean arterial blood pressure <60 mm Hg
- Decline of systolic blood pressure (SBP) or mean arterial pressure (MAP) by 20% from baseline

Presentation

- Chest pain
- Short of breath
- Nausea
- Altered mental status
- Oliguria

Pathophysiology

The pathophysiology is often multifactorial. Anything that decreases preload, afterload, or contractility can cause hypotension.

Immediate Management

- Administer IV fluid.
- Check for medication error (e.g., drug overdose or swapped syringe).
- Administer ephedrine 5 mg IV bolus (may repeat as necessary).
- Administer phenylephrine 100 mcg IV bolus (may repeat as necessary).
- Epinephrine 5–10 mcg IV bolus (may repeat as necessary).

DIFFERENTIAL DIAGNOSIS

- Deep level of anesthesia
- Hypovolemia
- Vasodilation caused by spinal or epidural anesthesia
- Anaphylaxis
- Pericardial tamponade
- Pneumothorax
- Acute heart failure
- Endocrine dysfunction

Diagnostic Studies

- ECG
- Echocardiogram
- Intra-arterial catheter for continuous blood pressure (BP) monitoring

Subsequent Management

- Treat the underlying cause (e.g., fluid resuscitation, discontinue vasodilating drugs)

- If unresponsive to catecholamines, consider vasopressin 40- μ IV bolus
- Administer corticosteroids if adrenal insufficiency is suspected: hydrocortisone 100 mg IV in adults.

Risk Factors

- Dehydration
- Age >65 years
- Beta-blocker or calcium channel blocker therapy
- Neuraxial analgesia
- Cardiac disease
- Medications

Prevention

- Maintain normovolemia: Provide adequate fluid resuscitation during surgery.
- Monitor the level of spinal and epidural anesthetics.
- Administer vasodilating medications slowly (e.g., protamine, vancomycin).

Special Considerations

- Hypotension can be caused by a myriad of abnormalities. Rapid differential diagnosis can narrow the therapeutic options and improve patient outcome.

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 7.4: Monitoring and medications. *Circulation*. 2005; 112: IV-78–IV-83.

Myocardial Ischemia

Definition

Oxygen supply inadequate to meet myocardial demand.

Presentation

- Chest pain, pressure, or tightness often localized behind the sternum
- Radiates to either arm, neck, jaw, back or abdomen
- Usually associated with increase in either physical or emotional activity
- Dysrhythmias, ST segment changes, or hypotension may be the only findings in an anesthetized patient.

Pathophysiology

Myocardial ischemia may be caused by coronary obstruction (physical obstruction from plaque and/or thrombus, or physiologic from coronary spasms), decreased oxygen carrying capacity, or decreased coronary perfusion pressure.

Immediate Management

- Administer supplemental O₂ to maintain SpO₂ >90%.
- Administer oral nitroglycerine 0.4 mg sublingual or 0.5 mcg/kg/min IV (starting dose). Note: Nitroglycerine is contraindicated in patients with systolic pressure <90 mm Hg, diastolic pressure <30 mm Hg, or severe bradycardia.
- Administer morphine 1- to 2-mg IV boluses to control pain if the patient is awake.
- Administer beta-blockers (e.g., labetalol 5-mg IV bolus, esmolol 50 mcg/kg/min) to decrease heart rate and blood pressure. (Decrease myocardial oxygen demand.)
- Request an emergency cardiology consult for possible thrombolysis or stenting.
- Discuss anticoagulation with the surgical service. If safe:
 - Administer aspirin 325 mg chewed and swallowed.
 - Begin a heparin infusion or (60 U/kg bolus, 12 U/kg/h).
 - Administer a loading dose of clopidogrel 600 mg PO if possible.
 - Manage arrhythmias aggressively.
 - Consider fibrinolysis (tenecteplase within 30 minutes, only for ST segment elevation myocardial infarction [STEMI]).

DIFFERENTIAL DIAGNOSIS

- Heartburn/dyspepsia
- Myopathic pain
- Thoracic (bone and cartilage) or pleuritic pain

Diagnostic Studies

- ECG
- Echocardiogram

Subsequent Management

- Refer the patient for primary percutaneous coronary intervention (coronary angioplasty and/or stents).
- Percutaneous cardiopulmonary support (pacemaker, intra-aortic balloon pump) if the patient is hemodynamically stable.

- Prepare for emergency coronary artery bypass grafting (CABG) to preserve myocardium if the patient is unstable or percutaneous therapy is contraindicated or unavailable.
- Long-term care should include antiplatelet therapy (e.g., clopidogrel) to reduce mortality.

Risk Factors

- Age >50 years
- Males are at greater risk than females.
- Pre-existing hypertension
- Pre-existing diabetes mellitus
- Hyperlipidemia
- History of smoking
- Family history
- Illicit drug use (cocaine)

Prevention

Maintain normal hemodynamics and monitor at-risk patients carefully.

Special Considerations

- Mortality for emergency CABG for acute coronary syndrome (due to left main coronary) is approximately 9%.
- Although outcomes of emergency CABG intervention remain unclear for acute myocardial infarction (MI), cardiogenic shock prior to CABG intervention has been identified as a significant predictor of mortality.
- Despite concerns of excessive bleeding during CABG, clopidogrel is recommended as an early intervention (on admission), even for those who ultimately undergo CABG.

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 8: Stabilization of the patient with acute coronary syndromes. *Circulation* 2005; 112: IV-89–IV-110.

American Heart Association Task Force on Guidelines. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: executive summary. *J Am Coll Cardiol*. 2013; 61(4): 485–510.

Rao AK, Pratt C, Berke A, Jaffe A, Ockene I, Schreiber TL, Bell WR, Knatterud G, Robertson TL, Terrin ML Thrombolysis in Myocardial Infarction (TIMI) Trial—phase I: hemorrhagic manifestations. *J Am Coll Cardiol*. 1988; 11(1): 1–11.

Postoperative Hemorrhage

Definition

Excessive bleeding in the postoperative period (>300 mL/h in the first few hours after surgery).

Presentation

- Increased output from chest tubes or other drains
- Hemodynamic instability

Pathophysiology

Coagulopathy after cardiothoracic surgery may be a result of anti-coagulation therapy, resulting in chest wall bleeding. Inadequate surgical hemostasis resulting in suture line bleeds and elevated blood pressure causing generalized bleeding, are also contributory.

Immediate Management

- Immediately notify the surgical team if bleeding is >300 mL/h over the first few hours. The patient may require emergency surgery.
- Send blood for laboratory coagulation studies (e.g., prothrombin time [PT], international normalized ratio [INR], platelet count, fibrinogen level). Transfuse factors or platelets as necessary to correct.
- Correct hypothermia (may interfere with coagulation)
- Fluid resuscitate with crystalloid or blood products as necessary. Note: If the patient is rapidly bleeding, it may be necessary to activate the rapid transfusion protocol.

DIFFERENTIAL DIAGNOSIS

- Hypovolemia
- Cardiac dysfunction
- Pericardial tamponade

Diagnostic Studies

- Coagulation studies
- Temperature

Subsequent Management

- Blood component therapy
- Antifibrinolytic therapy

Risk Factors

- Elevated blood pressure
- Inadequate surgical hemostasis

- Coagulopathies
- Hypothermia

Prevention

- Adequate surgical hemostasis is the best prevention.
- Adequate reversal of heparin (with protamine) confirmed with activated clotting time (ACT) is mandatory. Adjuncts like fibrin glue at specific surgical sites can be useful.

Special Considerations

- If chest tube output decreases suddenly, carefully examine them for clots. Occluded chest tubes may cause cardiac tamponade. If the patient is requiring massive transfusion, follow the ionized calcium level and correct as necessary. Avoid hypothermia by warming all infusions. Consider using scopolamine, midazolam, or ketamine to sedate patients who are hypovolemic and hemodynamically unstable.

Further Reading

Despotis G, et al. Prediction and management of bleeding in cardiac surgery. *J Thromb Haemost.* 2009; 7(Suppl 1): 111–117.

Pulmonary Embolism

Definition

Material (e.g., thrombus, air, fat) that travels from a primary site (e.g., deep veins of the lower extremities for thrombus) and eventually becomes lodged in the pulmonary artery or its branches.

Presentation

- Minor pulmonary emboli may be undetected.
- Massive pulmonary emboli usually cause sudden, severe pulmonary and hemodynamic compromise, which most commonly includes tachycardia, tachypnea, and rales.
- Classic triad is dyspnea, hemoptysis, and chest pain (in an awake patient).
- Increased central venous pressure, coupled hypoxia, hypocapnea, and respiratory acidosis on ABG, especially when coupled with a sudden decrease in end-tidal CO_2 .
- Cyanosis

Pathophysiology

More than 95% of pulmonary thrombi are from lower extremity deep vein system. Sluggish blood flow combined with vein wall pathology and a hypercoagulable state are the usual etiologies.

Immediate Management

- If possible, begin anticoagulation (e.g., begin a heparin infusion. Administer heparin 5000 units IV bolus followed by an infusion of 1000 U/h).
- Thrombolytic therapy
- Hemodynamic support and resuscitation

DIFFERENTIAL DIAGNOSIS

- Acute myocardial infarction
- Severe bronchospasm
- Anaphylaxis
- Pneumothorax

Diagnostic Studies

- D-dimer assays (negative predictive value)
- Helical CT scan
- Echocardiography (to evaluate right ventricular dilation and strain)
- Evidence of right ventricular strain or right bundle branch block on ECG
- Ventilation perfusion scan
- Pulmonary angiogram

Subsequent Management

- Femoral-femoral bypass may be required for cardiopulmonary support
- Surgical embolectomy
- Inferior vena cava filter implantation

Risk Factors

- Virchow's triad:
 - Coagulation anomalies
 - Venous stasis of blood
 - Vein wall pathology

Prevention

- Deep venous thrombosis prophylaxis with anticoagulation and compressive stockings
- IVC filter

Special Considerations

- Surgical embolectomy has a high mortality.
- Brain natriuretic peptide elevation predicts right ventricular dysfunction and mortality.
- Elevated troponins are similarly associated with higher mortality.

Further Reading

DeLoughery TG. Venous thrombotic emergencies. *Emerg Med Clin North Am.* 2009; 27(3): 445–458.

Thoracic Aortic Dissection

Definition

Dissection of the intimal and medial layers of the thoracic aortic wall by penetrating blood. *Type A* dissections involve the ascending aorta, whereas *Type B* dissections involve the descending aorta.

Presentation

- Chest pain
- Back pain (often described as “ripping”)
- Congestive heart failure with aortic insufficiency
- Pericardial tamponade
- Elevated blood pressure
- Dyspnea and hoarseness caused by compression of the recurrent laryngeal nerve compression or trachea
- Hemoptysis caused by tracheal erosion
- Myocardial ischemia

Pathophysiology

Blood most commonly penetrates an intimal tear and separates the intima and media. This differs from an aneurysm, in which all three layers of the vessel wall are dilated. Tears in the ascending aorta and aortic arch make up 70% of all dissections. Vasa vasorum rupture has been implicated in a minority of cases without intimal tear.

Immediate Management

- Administer vasodilators to maintain systolic BP 105–115 mm Hg (e.g., nicardipine infusion starting at 5 mg/h or nitroprusside infusion starting at 0.25 mcg/kg/min).
- Administer beta-blockers (e.g., labetalol or esmolol infusion) to control heart rate. Goal is 60–80 bpm.

Immediate Management (*continued*)

- Transfuse packed red cells and/or coagulation factors as necessary.
- Refer the patient to a cardiothoracic surgeon for emergency repair.

DIFFERENTIAL DIAGNOSIS

- Acute myocardial infarction
- Cardiogenic shock
- Pancreatitis
- Thoracic outlet syndrome

Diagnostic Studies

- Chest X-ray (mediastinal width >8 cm, look for >5-mm space between aortic arch and the calcified aortic intima)
- Computed tomography scan of the chest
- Transesophageal echocardiography
- Angiogram

88

Subsequent Management

Continuous assessment of organ function is important. Neurologic changes, deteriorating kidney function, and gastrointestinal perfusion (which may manifest as metabolic acidosis) are all indications for acute surgical intervention.

Risk Factors

- Hypertension
- Age >60 years
- Males are at greater risk than females.
- Marfan syndrome and other connective tissue disorders
- Pregnancy

Prevention

Aggressive control of blood pressure prevents further injury.

Special Considerations

- Surgical mortality is >30%. Untreated 2-day mortality is 50% and 6-month mortality is as high as 90%.
- Preoperative pain medication should be used judiciously to permit evaluation of the patient's neurologic status. Neurologic deterioration may be the first sign of propagation of dissection into the neck.

- A balance between full-stomach precautions requiring rapid sequence induction and slow, controlled induction to maintain hemodynamic stability is required.
- Femoral artery cannulation may be required if the entire ascending aorta is involved, so as to allow for perfusion of the major vessels.
- Left radial artery is preferred for insertion of an intra-arterial catheter in patients with ascending aortic dissections because the right subclavian artery may be involved.

Further Reading

Kohl BA, McGarvey ML. Anesthesia and neurocerebral monitoring for aortic dissection. *Semin Thorac Cardiovasc Surg.* 2005; 17(3): 236–246.

Subramanian S, Roselli EE. Thoracic aortic dissection: long-term results of endovascular and open repair. *Semin Vasc Surg.* 2009; 22(2): 61–68.

Valvular Disease: Aortic Regurgitation

89

Definition

Abnormalities in the leaflets or supporting structures of the aortic valve, resulting in retrograde flow into the left ventricle during diastole.

Presentation

- Dyspnea
- Fatigue
- Palpitations
- Angina

Pathophysiology

Aortic insufficiency leads to increased LV systolic and diastolic volumes. A regurgitant fraction of <40% is well tolerated with minimal symptoms. As regurgitation approaches 60%, LV end-diastolic pressure (LVEDP) increases. LVEDP gradually increases indicating worsening dilation and hypertrophy of the LV and eventually leads to pulmonary edema.

Immediate Management

- Mildly vasodilate the patient to minimize afterload and promote forward flow (e.g., Begin a nicardipine infusion, start at 5 mg/h)
- Avoid anesthetic induction with ketamine (may increase afterload).
- Administer fluid to maintain preload.
- Increase heart rate (goal 90 bpm) to minimize diastolic time.

DIFFERENTIAL DIAGNOSIS

- Other causes of diastolic murmurs (e.g., mitral stenosis)
- Pulmonary edema
- Coronary artery disease
- Congestive heart failure

Diagnostic Studies

- ECG
- Echocardiogram
- Refer for coronary angiography.

Subsequent Management

- Decreased afterload with by administering vasodilators
- Refer for aortic valve repair or replacement.

Risk Factors

- Marfan syndrome
- Bacterial endocarditis
- Cystic medionecrosis
- Trauma
- Aortic dissection
- Bicuspid aortic valve

Prevention

Early detection and medical management of symptoms.

Special Considerations

Regurgitant fraction is calculated as

$$\text{Regurgitant Fraction} = \frac{\text{Backward flow}}{\text{Total Flow}}$$

Total flow = (End diastolic volume – End systolic volume) × HR

Forward flow = Cardiac output

Backward flow = Total flow – forward flow

An intra-aortic balloon pump is contraindicated in patients with significant aortic insufficiency.

Further Reading

Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP 3rd, Guyton RA, O’Gara PT, Ruiz CE, Skubas NJ, Sorajja P, Sundt TM 3rd, Thomas JD; American College of Cardiology/American Heart Association Task Force on Practice Guidelines. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: executive summary: a report of

the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol.* 2014; 63(22): 2438–2488.

Valvular Disease: Aortic Stenosis (AS)

Definition

Narrowing of the aortic valve diameter at the valvular, subvalvular or supravalvular level. Normal valve area is 2.6–3.5 cm². Aortic stenosis is graded as mild (1.5–2.5 cm²), moderate (1.0–1.4 cm²), severe (0.6–0.9 cm²), or critical (<0.6 cm²).

Presentation

- Angina
- Syncope
- Dyspnea

Pathophysiology

A congenital bicuspid valve is the major predisposing factor (>75% of cases) followed by rheumatic fever and senile calcific degeneration.

Mild aortic stenosis leads to left ventricular hypertrophy and increased left ventricular systolic and end-diastolic pressures to compensate for increased resistance to flow. Progression of left ventricular dilation and hypertrophy further increases left ventricular end diastolic volume and increase workload. This eventually leads to pulmonary edema and increases the risk of sudden death.

91

Immediate Management

- Treat hypotension immediately with a vasoconstrictor (e.g., phenylephrine 100-mcg IV bolus).
- Maintain normovolemia.
- Avoid drugs that decrease systemic vascular resistance.
- If necessary, begin a phenylephrine infusion to augment preload and maintain afterload (starting dose 0.5 mcg/kg/min).
- Decrease heart rate (goal is 50–70 bpm)
- Preserve sinus rhythm: Immediate cardioversion for supraventricular dysrhythmias

DIFFERENTIAL DIAGNOSIS

- Pulmonary hypertension
- Coronary artery disease
- Congestive heart failure
- Systolic murmurs (mitral regurgitation)

Diagnostic Studies

- ECG
- Echocardiogram
- Chest radiograph

Subsequent Management

- Afterload reduction is not helpful because the stenotic valve resists forward flow.
- Administer alpha-adrenergic agents to treat hypotension because they maintain systemic vascular resistance (SVR). A high SVR ensures that diastolic pressure is sufficient to perfuse the hypertrophied myocardium.
- Patients with aortic stenosis do not usually tolerate beta-blockade well because beta-blockers decrease myocardial contractility. They may be beneficial in a patient with idiopathic hypertrophic subaortic stenosis (which is a dynamic stenosis).
- Patients with critical AS should be monitored intraoperatively with with transesophageal echocardiography.

Risk Factors

- Bicuspid aortic valve
- History of rheumatic fever
- Age >70 years
- Males are at greater risk than females.

Prevention

Early detection and medical management of symptoms.

Special Considerations

- LV dysfunction (i.e., CHF) in patients with aortic stenosis is associated with high risk for sudden death.
- Myocardial hypertrophy is a risk factor for subendocardial ischemia. Myocardial ischemia may be difficult to detect because ECG signs of left ventricular hypertrophy may mask ischemic changes.

Further Reading

Frogel J, Galuska D. Anesthetic considerations for patients with advanced valvular heart disease undergoing noncardiac surgery. *Anesthesiol Clin*. 2010; 28(1): 67–85.

Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP 3rd, Guyton RA, O’Gara PT, Ruiz CE, Skubas NJ, Sorajja P, Sundt TM 3rd, Thomas JD; American College of Cardiology/American Heart Association Task Force on Practice Guidelines. 2014 AHA/ACC guideline for the management

of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol.* 2014; 63(22): 2438–2488.

Valvular Disease: Mitral Regurgitation (MR)

Definition

- Blood flowing backward from the left ventricle into the left atrium during systole. Regurgitant fraction = regurgitant volume/LV stroke volume
- Mild MR: Regurgitant fraction <30%
- Moderate MR: Regurgitant fraction 30%–60%
- Severe MR: Regurgitant fraction >60%

Presentation

- Fatigue
- Dyspnea
- Orthopnea

Pathophysiology

- Mitral regurgitation (MR) can be *primary* (due to a defective valve) or *secondary* (due to LV dilation). The most common cause of MR is mitral valve prolapse. Myocardial infarction with papillary muscle dysfunction may also present as acute onset MR.
- Acute MR leads to increased left atrial volumes and pressures, which are transmitted to the pulmonary circuit. Compensatory tachycardia maintains cardiac output at the expense of increased myocardial oxygen demand.
- Chronic, slow onset MR causes compensatory left ventricular hypertrophy and left atrial dilation. This maintains forward flow and normal pressures to the pulmonary circuit, and is responsible for the relative absence of symptoms. When the regurgitant fraction increases to 60%, the hypertrophic and dilated LV is unable to compensate and heart failure ensues.

Immediate Management

To ensure forward flow:

- Maintain preload
- Avoid bradycardia (increases LV and regurgitant volume)
- Decrease afterload (e.g., vasodilator such as hydralazine 10 mg IV)
- Avoid further increases in pulmonary vascular resistance (hypoxia, hypercapnea, acidosis)

DIFFERENTIAL DIAGNOSIS

- Primary pulmonary hypertension
- Myocardial ischemia
- Cardiomyopathy

Diagnostic Studies

- Echocardiogram
- Giant V wave on pulmonary artery occlusion pressure tracing

Subsequent Management

- Consider nitric oxide is a pulmonary vasodilator
- Consider prostaglandin E1
- Refer for surgical intervention

Risk Factors

- Mitral valve prolapse
- Myocardial ischemia with papillary muscle dysfunction
- Bacterial endocarditis

94

Prevention

Once LV dysfunction has become established, it may be irreversible; early intervention offers the best chance of a good outcome.

Special Considerations

- Atrial fibrillation occurs in approximately 75% of cases of MR.

Further Reading

Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP 3rd, Guyton RA, O’Gara PT, Ruiz CE, Skubas NJ, Sorajja P, Sundt TM 3rd, Thomas JD; American College of Cardiology/American Heart Association Task Force on Practice Guidelines. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol.* 2014; 63(22): 2438–2488.

Valvular Disease: Mitral Stenosis (MS)

Definition

- Normal valve area 4–6 cm² (valve index 4–4.5 cm²/m²)
- Symptomatic at 1.5–2.5 cm² (valve index 1–2 cm²/m²)
- Critical <1 cm²
- Incompatible with life <0.3 cm²

Presentation

- Dyspnea
- Fatigue
- Palpitations
- Paroxysmal nocturnal dyspnea
- Hemoptysis
- Angina

Pathophysiology

- Usually secondary to rheumatic heart disease, which causes fusion of valvular commissures and progressive scarring.
- Mild disease can be asymptomatic if physiologic compensation is adequate. Increased filling pressure in the left atrium with mild mitral stenosis is not usually transmitted to the pulmonary circulation. Gradually progression of stenosis increases pulmonary vascular resistance and right ventricle pressure. Hypertrophy and dilation of the RV further compromise LV function by shifting the interventricular septum toward the LV, decreasing LV volumes.

95

Immediate Management

- Maintain preload (increases can precipitate pulmonary edema)
- Slow heart rate (maximize time spent in diastole)
- Maintain pulmonary vascular resistance (avoid increases in pulmonary vascular resistance caused by hypoxia, hypercarbia, and acidosis)
- Maintain sinus rhythm: Treat arrhythmias immediately with DC cardioversion.

DIFFERENTIAL DIAGNOSIS

- Primary pulmonary hypertension
- Other causes of diastolic murmurs (e.g., aortic regurgitation)
- Myocardial ischemia

Diagnostic Studies

- Transesophageal echocardiogram
- Pulmonary artery catheter—large A wave on PA tracing

Subsequent Management

- Afterload reduction is not helpful because the proximal stenosis at the mitral valve is the limiting factor.
- AV pacing with long PR intervals (to allow adequate filling) may be necessary.

- Begin treatment with beta-blockers.
- Maintain sinus rhythm.
- Refer the patient for surgical intervention.

Risk Factors

- Females are at greater risk than males.
- History of rheumatic fever

Prevention

Early detection and medical management of symptoms and early intervention to avoid irreversible ventricular dysfunction.

Special Considerations

- Atrial contraction contributes to the LV stroke volume and is extremely important because it could be as high as 30% of the stroke volume.
- Even though mitral commissurotomy results in a restenosis rate of 30% in 5 years, early morbidity is lower than that of valve replacement.

Further Reading

Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP 3rd, Guyton RA, O’Gara PT, Ruiz CE, Skubas NJ, Sorajja P, Sundt TM 3rd, Thomas JD; American College of Cardiology/American Heart Association Task Force on Practice Guidelines. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol.* 2014; 63(22): 2438–2488.

Venous Gas Embolism

Definition

Gas bubbles in the blood stream (usually in the venous system) that interrupt blood flow.

Presentation

- In an awake patient, the earliest signs include coughing and chest pain.
- Arterial desaturation
- Decreased end-tidal CO₂
- If systemic embolization occurs, ischemic changes may be seen on the ECG.
- In awake patients, acute neurologic deficits may be noted.
- The surgeon may note bubbles in arterial blood.

- If a large volume of gas is rapidly entrained, cardiovascular collapse will occur.

Pathophysiology

Two components are required for a gas embolism to occur: an interface between the vascular system and air, and a pressure gradient between air and the vascular system. Gas bubbles move to the pulmonary circulation and cause progressive right heart failure. They may also accumulate in the right ventricular outflow tract, decreasing cardiac output. Gas emboli can also cause ischemic injury in end organs if the bubbles pass into the arterial circulation through a right-to-left shunt.

Immediate Management

- Immediately alert the surgeon. Ask him or her to flood the field with irrigation fluid or cover it with wet sponges. He or she should also find and occlude the point of gas entrainment using bone wax and/or electrocautery.
- Call for help.
- Increase FiO_2 to 100%.
- If possible, position the OR table flat or head-down to place the surgical wound below the level of the right atrium.
- Support the blood pressure: Administer vasoactive drugs to maintain a normal systolic blood pressure (e.g., phenylephrine 100 mcg IV bolus).
- Volume resuscitate with isotonic crystalloid or colloid solutions to support systemic blood pressure and increase right atrial pressure.
- If a central venous catheter is in place, it may be possible to remove air in the right atrium by aspirating from the most distal port.
- If cardiovascular collapse occurs, perform cardiopulmonary resuscitation with the patient in head-down and rolled-to-the-left lateral decubitus position.

DIFFERENTIAL DIAGNOSIS

- Pulmonary thromboembolism
- Stroke
- Myocardial ischemia
- Cardiogenic shock

Diagnostic Studies

- Sudden decrease in end-tidal CO_2
- Echocardiogram (most sensitive) shows gas bubbles in the right atrium.

- Precordial Doppler (sensitive but not specific for clinically significant emboli)
- Arterial blood gas reveals respiratory acidosis, increased PaCO_2 , hypoxia.

Subsequent Management

- In the case of massive embolism, or if gas has migrated to the arterial circulation, consider hyperbaric therapy.

Risk Factors

- Lung barotrauma
- Craniotomy in the sitting position (classic risk factor)
- Any surgical procedure in which the operative site is located above the level of the heart, including caesarean section, arthroscopic surgery of the shoulder, and total hip arthroplasty
- Laparoscopic surgery: Direct injection of CO_2 into the circulation through a trocar that is placed into a vein, or gas that enters veins at the operative site. Carbon dioxide bubbles are more soluble and disappear more quickly than nitrogen or oxygen, but can cause a symptomatic gas embolus.

Prevention

Maintain normovolemia. Low venous pressure is a risk factor for venous air embolism (VAE). Position the patient in such a way as to reduce the pressure gradient between operative site and the right atrium during surgery and also when inserting or removing a central venous catheter.

Special Considerations

- Twenty to thirty percent of the population have an asymptomatic patent foramen ovale, which allows gas bubbles in the venous circulation to move into the arterial circulation. This can result in stroke, myocardial ischemia, and other life-threatening complications.

Further Reading

Mirski M, et al. Diagnosis and treatment of vascular air embolism. *Anesthesiology*. 2007; 106(1): 164–177.

Chapter 5

Thoracic Emergencies

Peter S. Burrage, Marc S. Azran, and Michael Nurok

Bronchopleural Fistula	100
Cardiac Herniation after Pneumonectomy	103
Inhaled Foreign Body (Adult)	105
Intrathoracic and Mediastinal Lesions Causing Tracheal, Bronchial, Cardiac, and/or Vascular Obstruction	108
Major Hemorrhage during Mediastinoscopy	112
Pathophysiology	112
Special Considerations	114
One-Lung Ventilation: Hypoxemia	115
One-Lung Ventilation: Increased Airway Pressure	117
Tension Pneumothorax	119
Tracheal Injury	121

Bronchopleural Fistula

Definition

A communication between the bronchial tree and pleural space that causes a pneumothorax. A bronchopleural fistula (BPF) can be associated with pulmonary resection as well as with chronic infection such as tuberculosis or empyema. After resection, BPF can occur immediately postoperatively ("stump blow out") or in a more delayed fashion, typically presenting 1–2 weeks later. It may also be caused by penetrating chest trauma or iatrogenic injury such as central line insertion, barotrauma, or volutrauma caused by mechanical ventilation.

Presentation

- Inability to ventilate (complete stump blowout)
- Dyspnea
- Oxygen desaturation
- Increased inspiratory pressures
- Cyanosis
- Hypotension
- Tachycardia

DIFFERENTIAL DIAGNOSIS

- Endotracheal tube malposition
- Bronchospasm
- Extrinsic lung compression by a mass or fluid
- Hemothorax
- Cardiac tamponade

Immediate Management

- Immediate postoperative stump blowout:
 - Isolate the lungs with a double-lumen endotracheal tube or bronchial blocker (see page 407).
 - Insert a thoracostomy tube.
 - Schedule the patient for emergency surgical re-exploration.
- Delayed bronchopleural fistula
 - Insert a thoracostomy.
 - A bronchopleural fistula may permit airflow up to 16 liters/min (LPM) across the defect. A thoracostomy tube with an internal diameter of at least 6 mm is required to evacuate air rapidly enough to avoid tension pneumothorax.
 - High volume thoracostomy tube drainage system ideally capable of evacuating up to 35 LPM with a suction pressure of -20 cm H_2O .

Diagnostic Studies

- Chest radiograph after chest tube insertion on the affected side.
- A CT scan may demonstrate an air fluid level.

Subsequent Management

- Treat the underlying lung pathology and wean mechanical ventilation. Positive pressure ventilation creates a gradient between the airways and pleural space. This allows air to pass through the fistula and prevents it from closing. Minimizing the volume of the leak during mechanical ventilation promotes healing.
- Conventional mechanical ventilation
 - Minimize alveolar distension and airway pressure.
 - Use the lowest allowable minute ventilation (both rate and tidal volume).
 - Decrease inspiratory time by altering the I:E ratio or by increasing inspiratory flow rates.
 - Conduct regular spontaneous breathing trials with a view to the earliest possible ventilator liberation.
 - Use weaning modes to encourage spontaneous ventilation.
 - Use the least amount of positive end-expiratory pressure (PEEP) that is feasible.
 - Decrease the amount of chest tube suction as tolerated.
 - Treat bronchospasm and other airflow obstruction aggressively.
- Alternative modes of ventilation may decrease mean airway pressures.
 - Occlusion of the chest tubes during inspiration or adding PEEP to the chest tube system may minimize the egress of air through the fistula during ventilation; however, creation of a tension pneumothorax is a risk.
 - Placing the patient into the lateral decubitus position with the BPF in the dependent position theoretically decreases airflow through the fistula.
 - Consider differential lung ventilation through a double-lumen endotracheal tube. The normal lung may be ventilated conventionally, whereas the injured lung may be managed with any of the preceding techniques. Two synchronized ventilators are required.
 - High-frequency jet ventilation may be used to maintain oxygenation, but respiratory acidosis may occur because CO_2 elimination is decreased.

Invasive Management

- Bronchoscopy may be used to localize and seal a proximal small BPF with fibrin, autologous blood, or cautery.
- A distal BPF that cannot be visualized may be occluded with a balloon.
- Chest tube or thoracoscopic sclerosis may incite an inflammatory response, thereby sealing a BPF.
- Surgical pleurodesis or bronchial stapling should be considered in refractory cases.
- Alert the surgical team if the patient requires 100% O₂ because electrocautery may cause a fire.

Risk Factors

- High-risk pulmonary surgery or lung volume reduction surgery
- Pulmonary infection
- Central line placement
- Excessive tidal volumes (>10 mL/kg) in a patient with lung injury
- Obstructive lung disease with auto-PEEP

Prevention

Decrease tidal volumes to 6 mL/kg

- Permissive hypercapnia
- Reduce inspiratory pressures.
- Monitor static respiratory compliance while increasing PEEP. Decrease PEEP if compliance falls.
- Wean the patient from mechanical ventilation early.
- Institute measures to avoid nosocomial pneumonia.
- Suspend mechanical ventilation while inserting a needle into the internal jugular or subclavian veins.
- Surgical reinforcement of stumps with a flap (e.g., intercostal muscle, omental tissue)

Further Reading

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Cardiac Herniation after Pneumonectomy

Definition

Herniation of the myocardium into an empty hemithorax through a pericardial defect created during pneumonectomy. Both left- and right-sided cardiac herniation may occur, causing severe hemodynamic instability. Mortality is 50% if recognized and 100% if left untreated.

Presentation

- Usually occurs during patient transport. May occur within the first 24 hours after surgery.
- Hypotension
- Tachycardia
- Cardiovascular collapse
- Dysrhythmias
- Electrocardiogram (ECG) changes (ST segment or axis changes)
- Right-sided herniation
 - Jugular venous distension
 - Cyanosis of the face
 - Absence of a left-sided cardiac impulse

Pathophysiology

- Right-sided cardiac herniation causes cardiac malposition.
 - Torsion of the cavoatrial junction severely impedes venous return.
 - Torsion of the great vessels or ventricular outflow tract obstructs blood flow.
 - The end result is myocardial ischemia and cardiovascular collapse.
- Left-sided cardiac herniation involves prolapse of the ventricles through the defect.
 - Cardiac orientation and venous return is preserved.
 - Strangulation of the myocardium and epicardial vessels causes myocardial ischemia.
 - Ventricular outflow tract obstruction also may occur.

DIFFERENTIAL DIAGNOSIS

- Myocardial infarction
- Hypovolemic shock
- Contralateral pneumothorax
- Pulmonary embolus
- Cardiac tamponade

Immediate Management

- Clinical scenario can help to differentiate between cardiac herniation (which requires immediate surgical intervention) and hypotension due to hypovolemia or deep anesthesia (which responds to fluid administration and/or vasopressors).

Management of Presumed Cardiac Herniation

- Alert the surgeon immediately.
- Reposition the patient with the operative side up.
- Decrease tidal volume and PEEP.
- Begin aggressive fluid resuscitation to correct relative preload deficiency.
- Support the blood pressure as necessary with vasopressors. Severe hypotension may require aggressive treatment with an infusion of phenylephrine (0.5–1 mcg/kg/min) or epinephrine (0.03–0.05 mcg/kg/min).
- If complete cardiac arrest occurs, CPR may make herniation worse, which underscores the importance of immediate surgical intervention and helps explain the high mortality rate of this complication.
- Surgical correction with reduction of myocardial prolapse and pericardial patch construction.

Diagnostic Studies

- This is a clinical diagnosis. Do not delay management for diagnostic studies.
- Chest radiograph
 - Right-sided herniation
 - Opacification of the right hemithorax from mediastinal shift
 - Abnormal (clockwise) configuration of a pulmonary artery catheter
 - Left-sided herniation may show a rounded opacity in the lower left hemithorax due to ventricular strangulation.
- Echocardiography (TTE or TEE) may reveal prolapsed myocardium or a malpositioned heart.

Risk Factors

- Pericardial defect of any size and an empty hemithorax
- Chest tubes placed to suction or water seal
- Positive pressure ventilation and PEEP
- Coughing caused by suctioning or extubation

Risk Factors (continued)

- Position changes in the operating room or during transport from the operating room (especially when the operative side is down).

Prevention

The surgeons should use a pericardial sling or patch close large pericardial defects. Primary closure of the pericardium has been used for smaller defects, although herniation can occur from a ruptured suture line.

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Inhaled Foreign Body (Adult)**Definition**

Aspiration of organic or inorganic material into the tracheobronchial tree.

Presentation

- At the laryngeal inlet: coughing, choking, hoarseness, or cyanosis
- Below the cords: inspiratory stridor and coughing
- In a bronchus: unilateral wheezing and coughing
- If diagnosis is delayed, the patient may present with recurrent pneumonia, empyema, hemoptysis, or bronchopleural fistula.

Pathophysiology

Organic material (e.g., beans or other food products) may absorb fluid from surrounding structures and become engorged, causing complete airway obstruction. Inspissated oral secretions and/or mucous plugs may also lead to respiratory arrest through an obstructive mechanism. Inorganic material that does not cause a significant obstruction may be tolerated for years with minimal symptoms.

DIFFERENTIAL DIAGNOSIS

- Pneumothorax
- Bronchospasm
- Tracheal injury
- Airway compression from the intrathoracic mass

Immediate Management

- In adults, foreign body aspiration is rarely a true emergency.
 - Monitor closely and give supplemental oxygen while the operating room is prepared.
 - The ability to convert from flexible to rigid bronchoscopy to thoracotomy should be immediately available.
- Emergency foreign body asphyxiation usually involves material at the supraglottic larynx or subglottic trachea.
 - Direct laryngoscopy can be performed while the patient is awake and the foreign body is removed with Magill forceps.
 - Intubation with an ETT may be used to push the object into a distal bronchus, enabling life-saving ventilation while the patient is prepared for bronchoscopy.

Diagnostic Studies

- Chest radiographs are useful only in patients with radio-opaque foreign bodies. Findings may include air trapping, atelectasis, pulmonary infiltrates, or mediastinal shift.

Subsequent Management

- *Rigid bronchoscopy* is the most effective therapeutic intervention and usually requires general anesthesia.
 - Positive pressure ventilation can wedge the foreign body distally, creating a ball-valve obstruction.
 - Spontaneous ventilation using an inhaled anesthetic is the preferred technique. Spontaneous breathing enables ventilation when the bronchoscopist's ocular window is open.
 - If neuromuscular blockade is needed to facilitate laryngoscopy, short-acting agents are preferred. Intermittent ventilation is possible through the bronchoscope. If positive pressure ventilation is necessary, consider an intravenous anesthetic technique to reduce operating room pollution with volatile agents.
- *Flexible bronchoscopy* is the tool of choice when the foreign body is wedged in a distal segment, in patients with cervical spine pathology, or when the patient is intubated and mechanically ventilated.

- Avoids general anesthesia and preserves spontaneous ventilation.
- Preservation of the cough reflex is imperative in patients with a full stomach and also aids in foreign body expulsion. Use local anesthetics sparingly.
- Sedatives should be administered judiciously.
- A laryngeal mask airway may be inserted in an awake patient after topical anesthesia of the oropharynx, and can be used as a conduit for the flexible bronchoscope. This provides some degree of airway control and is large enough to allow for extraction of the foreign body.
- Special forceps and snares are passed through the working port of the bronchoscope. The bronchoscope, grasping device, and foreign body are all removed as a unit. If the object dislodges in the proximal trachea, place the patient into the Trendelenburg position, and ask him or her to cough.
- If an endotracheal tube (ETT) is in place, the object may not pass through the ETT. In this case, it may be necessary to remove the endotracheal tube along with foreign body while being grasped under visualization through a bronchoscope.

Risk Factors

- Advanced age
- Neurologic disorders with impaired swallowing
- Alcohol and sedative use
- Trauma with loss of consciousness
- General anesthesia
- Seizures

Special Considerations

- Massive hemoptysis may occur when the foreign body is removed. Consider lung isolation with a double-lumen ETT or bronchial blocker if bleeding cannot be controlled. Preparations should be made for thoracotomy or bronchial artery embolization.
- Prolonged rigid bronchoscopy may lead to postoperative laryngeal edema. Treatment options include:
 - Nebulized racemic epinephrine
 - Helium/oxygen mixtures
 - Dexamethasone 10-mg IV bolus
- Failed bronchoscopy may ultimately require thoracotomy and bronchotomy to remove the impacted object.

Further Reading

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Intrathoracic and Mediastinal Lesions Causing Tracheal, Bronchial, Cardiac, and/or Vascular Obstruction

Definition

Extrinsic or intrinsic obstruction of intrathoracic structures caused by tumor, tracheal disease, or vascular compression, with potential to lead to cardiovascular collapse and/or the complete inability to oxygenate and ventilate.

Presentation

- Dyspnea
- Cough
- Orthopnea
- Inspiratory stridor or expiratory wheeze
- Hoarseness
- Hemoptysis
- Obstructive pneumonia

Pathophysiology

The common causes of obstruction include:

- Malignancy
- Primary airway tumors (usually intrinsic): squamous cell carcinoma, adenoid cystic carcinoma, or carcinoid tumor
- Primary adjacent tumor (usually intrinsic): anterior or middle mediastinal tumors, lung carcinoma, or esophageal carcinoma
- Metastatic cancer (extrinsic): renal, breast, colon, or sarcoma
- Tracheal disease related to stenosis or tracheomalacia (intrinsic)
- Vascular compression or ring (extrinsic)

General anesthesia can cause cessation of spontaneous ventilation, increased intrapleural pressure, and decreased functional residual capacity. As a result, airways that had been patent can collapse and cause worsening or complete obstruction.

DIFFERENTIAL DIAGNOSIS

- Foreign body aspiration
- Bronchospasm
- Pneumothorax

Immediate Management

Pre-procedure

- Identify the location of the lesion on imaging studies.
- Identify the relationship of the lesion to adjacent structures.
- Anticipate the potential for compression of the trachea, bronchi, and cardiovascular structures (>50% compression by tracheal lesions on imaging warrants a conservative approach).
- Insert an intra-arterial catheter if there is a risk of cardiovascular compromise.
- Obtain large-bore IV access. If superior vena cava (SVC) involvement or syndrome is suspected, insert a large-bore intravenous (IV) line in a lower extremity.
- Ensure IV fluids can be administered rapidly.
- Ensure that vasopressors are immediately available.
- Equipment and personnel skilled in rigid bronchoscopy should be immediately available.
- The surgical team must be present in the room.
- Femoral arterial and venous access is established, with standby extracorporeal support device in cases in which the likelihood of an inability to ventilate is high (e.g., for large compressive lesions in a patient unable to lie flat without symptoms).

During the Procedure

- A local anesthetic technique in the awake, spontaneously breathing patient is safest in patients with severe or potentially severe obstruction.
- Avoid sedatives, or, if they are necessary, administer incrementally in small doses. Consider using short-acting drugs (e.g., propofol, remifentanyl) or drugs that can easily be reversed.
- Maintain the patient in the sitting position if possible.
- Stepwise airway approach:
 - Awake fiberoptic examination of airway, trachea, and bronchi by anesthesia and surgical team to characterize the

Immediate Management (*continued*)

lesion and plan an approach to definitive airway management (may be facilitated by the use of a laryngeal mask airway—maintaining patient awake throughout—following topical local anesthesia)

- Awake fiberoptic intubation with passage of tube distal to lesion if possible. If this is not possible, consider alternative strategies for securing the airway while maintaining spontaneous ventilation, including the use of extracorporeal support.
- Securing the airway *after* induction should be attempted only with great caution, and with appropriately skilled individuals immediately available.

Induction:

- The airway is most safely managed *prior* to the induction of general anesthesia.
- Gradually administer an inhalational agent or small doses of intravenous agents while maintaining spontaneous ventilation.
- Attempt to assist with bag-ventilation
- If successful
 - Overtake spontaneous ventilation with positive pressure.
 - Gradually increase the depth of anesthesia.
- If not successful
 - Shifting the lesion by placing the patient in the lateral decubitus or prone position may improve ventilation.
 - Awaken the patient and reconsider the airway approach and/or using extracorporeal support.
- If neuromuscular blockade is required, use a small dose of succinylcholine *after* the airway is secure and the ability to provide positive pressure ventilation has been confirmed. **Neuromuscular blockade may cause the airway to collapse in a marginal patient.** If succinylcholine is tolerated, longer-acting agents can be administered safely.
- If the airway becomes completely obstructed, and is not relieved by repositioning the patient (lateral decubitus or prone position), attempt to pass a rigid bronchoscope or an armored endotracheal tube past the obstruction. An alternative rescue strategy is to pass a jet ventilator cannula distal to the lesion.
- An intravenous anesthetic technique is preferable for maintenance, as inhalational agents will contaminate the operating room with surgical manipulation of the airway.

Diagnostic Studies

- Neck and chest radiographs may demonstrate tracheal deviation, endoluminal narrowing, and obstructive pneumonia.
- Computed tomography (CT) imaging of the neck and chest can determine the exact location, length, and nature of the obstruction.
- Magnetic resonance imaging (MRI) and CT angiography are useful in characterizing vascular malformations.
- Transthoracic echocardiography may be useful in evaluating the pericardium for malignant effusion or tumor.
- Flow-volume loops are neither sensitive nor specific in characterizing obstruction.

Subsequent Management

- Surgical airway resection and reconstruction
- Tracheal (or bronchial) stent placement for symptomatic relief.
- Awake tracheostomy with a long-length prosthesis allows for control of the airway distal to the lesion.
- Endobronchial brachytherapy or external beam radiotherapy
- Preoperative radiation can significantly reduce tumor burden.
- Nd:YAG laser therapy in conjunction with rigid bronchoscopy can vaporize lesions and achieve hemostasis. Tracheal perforation, tracheal hemorrhage, and airway fire are risks with this procedure.
- Photodynamic therapy
 - IV photosensitizer is administered and retained in tumor cells.
 - Specific wavelength light activates the agent and generates cytotoxic oxygen radicals.

Risk Factors

- Prolonged intubation or tracheostomy may lead to stenosis or tracheomalacia.

Prevention

Daily spontaneous breathing trials and sedation holidays may reduce the incidence of complications from prolonged intubation.

Special Considerations

- There is a risk of acute intraoperative airway obstruction if the patient develops positional dyspnea while supine. If it may not be possible to maintain a patent airway during airway management or the surgical procedure, consider cannulating the

femoral artery and vein to permit extracorporeal oxygenation and ventilation.

- *Superior vena cava syndrome* is defined as obstruction of the SVC by tumor burden. These patients may have significant upper airway edema and friable tissue and require lower extremity IV access.
- Postoperative airway obstruction may occur if manipulation causes tumor swelling.
- Tracheal narrowing to <50% of normal confers a sevenfold increased risk.

Further Reading

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Major Hemorrhage during Mediastinoscopy

Definition

Bleeding during mediastinoscopy of >500 cc, or requiring exploration through a median sternotomy or thoracotomy. The incidence is 0%–0.4% in most large case series.

Presentation

- Arterial or venous surgical bleeding
- Hypotension
- Tachycardia
- Cardiovascular collapse

Pathophysiology

The surgical approach for cervical mediastinoscopy involves passage between the trachea and paratracheal fascia with dissection and biopsy of lymph nodes in the superior mediastinum. This region contains a number of anatomic structures in addition to the trachea, including the recurrent laryngeal nerve, thoracic duct, and esophagus, as well as major vasculature such as the azygous vein, innominate artery and vein, pulmonary artery, SVC, and aorta. Injury to these structures is uncommon, but may occur during surgical exploration of the mediastinum.

DIFFERENTIAL DIAGNOSIS

- Accidental biopsy of a vascular structure
- Azygous vein injury
- Innominate vein or artery injury
- Pulmonary artery injury
- Injury to the aortic arch

Immediate Management

- Tamponade bleeding via surgical compression (packing wound with gauze soaked in dilute epinephrine, digital pressure, compression with mediastinoscope).
- Establish large-bore IV access in the *lower extremities* if innominate vein injury is suspected.
- Begin volume resuscitation.
- Obtain cross-matched blood and set up rapid infusers.
- Insert an intra-arterial catheter for invasive blood pressure monitoring.
- Treat hypotension with ephedrine (5 mg IV) or phenylephrine (100 mcg IV) boluses. If refractory, consider phenylephrine infusion (0.5–1 mcg/kg/min).

Subsequent Management

- Surgical exploration and repair is the definitive treatment for refractory bleeding. The surgical approach will depend on the injured vessel.
 - **Midline sternotomy:** Innominate vein or artery, pulmonary artery, anterior SVC
 - **Right posterolateral thoracotomy:** azygous vein, right pulmonary artery, posterior SVC, or bronchial artery
- Lung isolation may enhance surgical exposure for right thoracotomy.

Previously easy intubation and bleeding is easily controlled by the surgeon:

- Confirm adequate neuromuscular blockade.
- Change the ETT to a left-sided double-lumen tube (with or without an airway exchange catheter).

Previously difficult intubation or uncontrolled bleeding:

- Intubate the left mainstem by advancing the existing endotracheal tube over a fiberoptic bronchoscope.
- Alternatively, insert a bronchial blocker into the right mainstem bronchus (see page 407).

- If definitive control at the bleeding source cannot be achieved despite a second incision for exposure, circulatory arrest with cardiopulmonary bypass may be required.

Risk Factors

- Aberrant blood vessels
- Superior vena cava (SVC) syndrome causing engorged vasculature
- Mediastinal inflammation as a result of prior chemotherapy, radiation therapy, or surgical procedure

Prevention

- Surgical palpation of the lesion or needle aspiration before biopsy to identify a blood vessel.
- Adequate neuromuscular blockade should be confirmed before biopsy to prevent movement during this critical period.

Special Considerations

- Persistent hemodynamic instability with only minor apparent blood loss may be due to cardiac tamponade. For example, post-biopsy bleeding from the bronchial artery into the pericardial sac causing tamponade has been described. Transesophageal echocardiography should be performed to rule out pericardial effusion and tamponade physiology.
- Patients who have had a prior sternotomy can present a particular challenge, especially if the vascular injury is at the level of the innominate vessels or the pulmonary artery. Prior sternotomy increases the risk for cardiac injury on repeat sternotomy; however, this must be weighed against the risk of uncontrolled bleeding from the injured vessel. The surgical team may consider a judicious upper hemisternotomy or resection.

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One-Lung Ventilation: Hypoxemia

Definition

Low PaO_2 and low SaO_2 in a patient who is receiving one-lung ventilation.

Presentation

- Low oxygen saturation by pulse oximetry
- Dark arterial blood
- Cyanotic patient
- Cardiac dysrhythmias

Pathophysiology

Pathophysiology is multifactorial. A shunt develops in the nonventilated lung after residual oxygen is resorbed. In the ventilated lung, regions with a low ventilation to perfusion ratio (West zone III) develop as a result of atelectasis and from compression by the nonventilated lung, mediastinal structures, and the diaphragm. Other causes of hypoxemia from the ventilated lung include hypoxic pulmonary vasoconstriction (causing redistribution of blood to the nonventilated lung and increasing shunt), secretions, and double lumen tube or lung isolation device malposition.

DIFFERENTIAL DIAGNOSIS

- Increased metabolic rate for oxygen
- Decreased oxygen delivery (i.e., a low cardiac output state)

Immediate Management

- Eliminate causes proximal to the double lumen tube, including disconnection and ventilator failure.
- Mild hypoxemia ($\text{SpO}_2 > 90\%$):
 - Increase FiO_2 to 100%.
 - Examine the airway with a fiberoptic bronchoscope to ensure correct position of double lumen tube or lung isolation device.
 - Check the ventilated lung for obstruction or secretions—passage of suction catheter is more efficient than using the narrow port on fiberoptic bronchoscope.
 - Ensure adequate cardiac output and oxygen carrying capacity.
 - Recruit the ventilated lung. Follow with addition of PEEP if using low tidal volume ventilation (Note: This will only work

Immediate Management (continued)

- if the lung is being ventilated in a noncompliant region on the low end of its pressure volume curve.)
- Apply continuous positive airway pressure to nonventilated lung (start with 5 cm H₂O to avoid distention of operative lung).
 - Consider switching to a total intravenous anesthetic technique. Hypoxic pulmonary vasoconstriction is impaired by potent volatile anesthetics.
 - Consider increasing tidal volume to 6–10 cc/kg.
 - If there is no improvement or the patient is severely hypoxic (SpO₂ <90%):
 - Inform the surgeon and ventilate both lungs.
 - If there is no improvement, or the preceding steps are not possible, ask the surgeon to clamp the pulmonary artery if a pneumonectomy of nonventilated lung is planned.
 - If there is no improvement, consider high-frequency jet ventilation to the operative lung.
 - If there is no improvement, consider nitric oxide or almitrine (not available in the United States).
 - If no improvement, consider extracorporeal membrane oxygenation (ECMO).

Diagnostic Studies

- Arterial and mixed venous blood gas measurement
- Fiberoptic bronchoscopy

Risk Factors

- Patients with increased or normal ventilation and perfusion to the operative lung will have a larger shunt during one-lung ventilation.
- Right-sided operations (right lung normally receives 60% of blood flow)
- Low PaO₂ during two-lung ventilation
- Normal or high FEV1/FVC ratio

Prevention

- Maintain two-lung ventilation as long as possible.
- Ensure proper double lumen endotracheal tube or lung isolation device position after changing the patient's position.
- Ensure appropriate ventilator settings when beginning one-lung ventilation (in patients with normal lungs, pressure control ventilation, 4–6 cc/kg ideal body weight with 5–10 cm H₂O PEEP, respiratory rate 10–15/min, FiO₂ 0.5–0.8).

- Adapt ventilatory strategy to lung pathology.
- Avoid using a high concentration of potent volatile anesthetic agents, which blunt hypoxic pulmonary vasoconstriction.
- Avoid delivering a large tidal volume or high level of PEEP to the ventilated lung. This increases pulmonary vascular resistance and may shunt blood to the nonventilated lung.

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One-Lung Ventilation: Increased Airway Pressure

117

Definition

Increase in peak or plateau inspiratory airway pressure

Presentation

- Elevated airway pressures during volume-controlled mode of ventilation
- Low tidal volume during a pressure-controlled mode of ventilation
- Low blood pressure

Pathophysiology

The most common cause of elevated airway pressure during one-lung ventilation is malposition of the double lumen endotracheal tube or lung isolation device, which causes a larger volume of gas is displaced into a smaller portion of lung and increases pressures. Other causes include delivering an excessive tidal volume to one lung, secretions, bronchospasm, and tension pneumothorax.

DIFFERENTIAL DIAGNOSIS

- Double lumen tube or lung isolation device malposition
- Obstruction within a double lumen endotracheal tube (blood, secretions, foreign body)
- Delivering an excessive tidal volume to one lung
- Bronchospasm

- Tension pneumothorax
- Air trapping/auto-PEEP (allow patient to exhale fully)

Immediate Management

- Inspect the ventilator tubing and endotracheal tube for kinks.
- Disconnect the patient from the ventilator and allow to exhale fully to exclude air trapping/auto-PEEP.
- Manually ventilate the patient to evaluate lung compliance.
- If high airway pressure is accompanied by hypoxemia, resume two-lung ventilation.
- Perform fiberoptic bronchoscopy to ensure proper positioning of double lumen tube or lung isolation device.
- Pass a suction catheter through the lumen leading to the ventilated portion of the lung to eliminate obstruction.
- Ensure delivery of appropriate tidal volume and PEEP settings for one-lung ventilation (in patients with normal lungs, pressure control ventilation, 4–6 cc/kg ideal body weight with 5–10 cm H₂O PEEP, respiratory rate 10–15/min, FiO₂ 0.5–0.8).
- Address other causes individually.

Risk Factors

- Surgical manipulation of the airway
- Change in patient position
- Right-sided double lumen endotracheal tube: The right upper lobe orifice may become misaligned. This causes the entire tidal volume to be delivered to the right lower and middle lobes.
- Obstructive lung disease is a risk factor for air trapping (use long expiratory times).

Prevention

Confirm correct placement of the double lumen endotracheal tube or lung isolation device after changing the patient's position or after significant surgical manipulation.

Further Reading

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Tension Pneumothorax

Definition

Abnormal presence of gas in the pleural cavity with inability to escape, causing pressure on intrathoracic structures.

Presentation

- High peak airway pressure
- Decreased tidal volume
- Decreased SpO₂, SvO₂
- Hypotension
- Tachycardia
- Distension of neck veins
- Subcutaneous emphysema
- Contralateral tracheal deviation
- Hyperresonance of the affected chest
- Hyperexpansion of the affected chest
- Reduced breath sounds in the affected chest
- Compression of bronchi on fiberoptic inspection of affected side
- Elevation of the mediastinum in the surgical field

Pathophysiology

Gas passes through the lungs and accumulates in the pleural space as a result of high airway pressure, rupturing of a bleb, or through the chest wall. A one-way valve effect prevents gas from escaping the pleural space. Increasing pressure in the pleural cavity from accumulation of gas results in clinical symptoms by compressing intrathoracic structures, including the mediastinum, lung, and blood vessels.

DIFFERENTIAL DIAGNOSIS

- Hyperinflation of the ventilated lung with intrinsic PEEP (disconnect patient from ventilator circuit and allow exhalation)
- Double lumen tube or lung isolation device malposition
- Bronchospasm
- Extrinsic lung compression
- Hemothorax
- Cardiac tamponade

Immediate Management

- Inform surgical team.
- Resume two-lung ventilation.
- Increase FiO₂ to 1.0.

Immediate Management (*continued*)

- Consult the surgeon about the ability to access the lung from the surgical field:
 - Ask the surgeons if they can dissect a plane to the affected pleural space between the aorta and esophagus posteriorly, and the pericardium anteriorly.
 - Accessing this space will immediately decompress the pneumothorax.
- If the preceding steps are not possible, position the patient supine and decompress the affected side with a large-bore needle or a long 14-gauge IV catheter in the second intercostal space at the midclavicular line.
- After the pneumothorax has been relieved, insert a chest tube.
- If there is uncertainty about the diagnosis and the patient is hemodynamically stable, fiberoptic bronchoscopy may show compression of major bronchi and a chest radiograph will provide a definitive diagnosis.

Diagnostic Studies

- Chest X-ray (in a hemodynamically stable patient) reveals midline shift to the contralateral side and a dark hemithorax on the affected side (due to lung collapse).

Subsequent Management

- Tube thoracostomy

Risk Factors

- Elevated peak airway pressures
- Malposition of the double lumen endotracheal tube causing high airway pressure
- Obstructive lung disease
- Acute lung injury
- Pleural blebs
- Penetrating chest wall injury
- Recent central venous catheter insertion

Prevention

- Adjust ventilator settings to avoid elevated airway pressure.
- Ensure that a double lumen endotracheal tube or lung isolation device is correctly positioned.

Special Considerations

- Tension pneumothorax has been reported during one-lung ventilation in the absence of the classic signs of hypoxemia and hypotension.

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Tracheal Injury

Definition

Injury to any portion of the extrathoracic or intrathoracic trachea that may involve complete or partial disruption of the trachea. Mortality estimates vary widely depending upon the etiology.

Presentation

- Dyspnea
- Hoarseness or stridor
- Signs of external trauma
- Subcutaneous emphysema
- Pneumothorax, pneumomediastinum or pneumopericardium
- Cyanosis and oxygen desaturation
- Hemoptysis

Pathophysiology

- *Iatrogenic* causes include endotracheal intubation, use of tube exchange catheter, percutaneous dilational tracheostomy, and cricothyroidotomy, which often causes tearing of the posterior membranous trachea.
- *Blunt trauma*
 - Tracheal injury is frequently within 2 cm of the carina.
 - Chest trauma may cause complete transection or a posterior membranous tracheal tear.
 - Upper airway trauma may result in fractured laryngeal cartilages.
 - Flexion-extension injury may precipitate full laryngotracheal separation.
- *Penetrating* trauma mostly occurs to the cervical trachea, but one-third may affect the larynx.
- *Mucosal tears* are often self-limited, but through-and-through tears require surgical management.

DIFFERENTIAL DIAGNOSIS

- Pneumothorax
- Foreign body aspiration
- Pulmonary hemorrhage
- Bronchospasm

Immediate Management

Iatrogenic Injury

Iatrogenic trauma that results in small tears to the middle and upper third of the trachea may be managed conservatively.

- Advance the tracheostomy or endotracheal tube beyond the lesion using a fiberoptic bronchoscope.
- Inflate the cuff to eliminate airway pressure on the proximal tear.
- Long-term ventilation may be required to allow healing.

Airway Management of Blunt or Penetrating Trauma

Airway manipulation may quickly turn a stable situation into a life-threatening one by precipitating complete obstruction.

- *General guidelines*
 - Tracheostomy equipment and a skilled surgeon should be present.
 - Maintain spontaneous ventilation. Avoid intravenous sedatives and neuromuscular blockers until the airway is secure.
 - Positive pressure ventilation before a cuff has excluded the injury can worsen existing pneumothorax, pneumomediastinum, or air dissection around the airways.
 - Awake fiberoptic bronchoscopy is the diagnostic and interventional procedure of choice for determining the nature of the injury as well as securing the airway.
 - The goal is to inflate a cuffed airway device or cannula distal to tracheal disruption to permit positive pressure ventilation.
 - If 100% FiO₂ is required to maintain oxygenation, advise the surgical team. Concomitant use of electrocautery may cause a fire.
- *Cervical tracheal and upper airway lesions*
 - Patients with open tracheal disruptions may be oxygenated via facemask and cannulated with the aid of a bronchoscope. A jet ventilator cannula may also be used for oxygenation.
 - Rigid bronchoscopy may be diagnostic when blood in the airway precludes flexible bronchoscopy. An inhaled

Immediate Management (continued)

anesthetic technique with spontaneous ventilation can be used to facilitate this approach, but there is a possibility of aspiration of gastric contents. The clinical scenario should dictate the best anesthetic approach.

- With blunt laryngeal injuries, attempted conventional laryngoscopy and endotracheal intubation may fracture the cricoid cartilage or provoke complete transection. Avoid cricoid pressure. Awake oral fiberoptic intubation or awake tracheostomy distal to the lesion are safe airway management options.
- *Lower tracheal lesions*
 - Blunt chest trauma disrupts the trachea within several centimeters of the carina. Tracheostomy will not permit ventilation in this situation.
 - Flexible fiberoptic bronchoscopy is essential in diagnosing and safely crossing the lesion. Intubation is best achieved over the bronchoscope.
 - The ETT cuff should be placed distal to the lesion and proximal to the carina if possible. Alternatively, consider endobronchial intubation or jet ventilation through a catheter located distal to the lesion.

Diagnostic Studies

- Do not delay airway management for diagnostic studies.
- Radiographic findings may include cervical emphysema, pneumothorax, pneumomediastinum or air column disruption.
- Computed tomography is sensitive, but the patient may not be able to lie flat. Sedatives may cause respiratory arrest and should be avoided until the airway is secure.
- All patients with tracheal injury should undergo an esophagoscopy to rule out esophageal perforation.

Associated Pathology

- Neurologic trauma including cervical spine and closed head injury
- Related airway pathology
 - Maxillofacial trauma
 - Laryngotracheal hematoma and edema
 - Subcutaneous emphysema of upper airway and epiglottis
- Esophageal injury
- Vascular injury with associated hemodynamic instability

Subsequent Management

- The approach for high lesions is a collar incision. The mediastinal trachea is repaired via a right posterolateral thoracotomy. The left approach is sometimes used, so the surgeon should be consulted.
- One-lung ventilation may be required to facilitate surgical exposure via a thoracotomy approach.
- An airway isolation device or blocker may be used.
- Alternatively, bronchial intubation with a double lumen endotracheal tube may safely be achieved over a fiberoptic bronchoscope or airway exchange catheter of adequate length.
- The endotracheal tube may need to be pulled back to facilitate visualization of the surgical anastomosis. An armored endotracheal tube and sterile circuit should be used to facilitate patient ventilation across the surgical field.
- At the conclusion of surgery, bronchoscopy should be used to position the cuff of the ETT distal to the anastomosis.

Prevention

Iatrogenic injury during endotracheal intubation can be minimized by ensuring that the tip of a stylet does not protrude past the tip of an ETT.

Special Considerations

- If it is impossible to maintain a patent airway during instrumentation or other stages of the procedure, the femoral artery and vein should be cannulated to permit extracorporeal oxygenation and ventilation prior to proceeding.
- Cricothyroid pressure (Sellick's maneuver) is contraindicated if upper tracheal or laryngeal injury is suspected, because it can cause complete tracheal disruption.

Further Reading

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Chapter 6

Metabolic and Endocrine Emergencies

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Acidosis	126
Acute Adrenal Insufficiency	128
Alkalosis	131
Anaphylaxis	133
Diabetes Insipidus	135
Diabetic Ketoacidosis and Hyperosmolar	
Hyperglycemia	137
Hypercalcemia	140
Hypocalcemia	142
Hyperkalemia	144
Hypokalemia	146
Hypermagnesemia	148
Hypomagnesemia	150
Hypernatremia	151
Hyponatremia	153
Hypothermia	155
Malignant Hyperthermia	158
Myxedema Coma	160
Pheochromocytoma	162
Porphyria	164
Thyroid Storm	167
Transurethral Resection of the Prostate Syndrome	169

Acidosis

Definition

Blood pH <7.36 (normal 7.36–7.45). Acidosis can include respiratory acidosis ($\text{PaCO}_2 > 45$ mm Hg), metabolic acidosis (plasma bicarbonate <22 mEq/L or arterial base excess < -3 mEq/L) or mixed.

Presentation

- Laboratory result on arterial or venous blood gas analysis
- Mild acidosis is often asymptomatic.
- Severe cases can cause vasodilation, shock, hypotension resistant to catecholamines, myocardial depression, and cardiac arrhythmias.
- Respiratory acidosis can include signs of CO_2 retention and respiratory failure such as hypoxemia, narcosis, and cyanosis.
- Metabolic acidosis can be associated with compensatory hyperventilation (*Kussmaul breathing*).

Pathophysiology

Metabolic acidosis is caused by increased acid generation (e.g., lactic acidosis), loss of bicarbonate (e.g., severe diarrhea), decreased renal acid excretion or dilution (volume expansion with hyperchloremic fluids [e.g., 0.9% saline]). *Respiratory acidosis* is caused by an increase in PaCO_2 ; most commonly from inability to eliminate CO_2 caused either by respiratory failure or by increased CO_2 production (e.g., malignant hyperthermia).

DIFFERENTIAL DIAGNOSIS

- Laboratory error

Immediate Management

- Establish IV access
- Mild cases of acidosis may not need urgent treatment.
- Intubate the trachea and begin mechanical ventilation in the setting of severe acidosis (pH <7.2), especially respiratory acidosis, or in cases of respiratory distress, respiratory failure, or hypoxemia.
- In ventilated patients, adjust minute ventilation to decrease PaCO_2 (10 mm Hg decrease in PaCO_2 increases pH by approximately 0.08).
- Severe metabolic acidosis (pH <7.2) can be treated with sodium bicarbonate (NaHCO_3). NaHCO_3 (mEq) = $0.5 \times \text{weight (kg)} \times (24 - \text{HCO}_3^- [\text{mEq/L}])$. Administer half of

Immediate Management (*continued*)

the calculated dose and repeat the ABG in 30 minutes to determine the need for additional therapy.

- Restore normal circulating blood volume. Lactated Ringer's solution is preferred to normal saline because it has a lower chloride content and reduces the risk of hyperchloremic acidosis.

Diagnostic Studies

- Basic metabolic panel
- Arterial blood gas analysis
- Calculation of anion gap

$$AG = ([Na^+] + [K^+]) - ([Cl^-] + [HCO_3^-])$$

Subsequent Management

- Diagnose and treat the underlying etiology of the acidosis.
- Respiratory acidosis due to respiratory failure should be treated by supporting ventilation, with either noninvasive or invasive means and by treating the underlying cause.
- Metabolic acidosis can be categorized as either high anion gap or normal (hyperchloremic) anion gap. Therapy should be directed to the cause(s) of the increased acid generation, loss of bicarbonate, or the diminished renal acid excretion.
- Renal replacement therapy may be indicated in patients with kidney failure and severe metabolic acidosis or in patients with severe acidosis due to toxin ingestion.

Risk Factors

- Respiratory failure
- Acute illness
- Acute blood loss
- Shock (hypoperfusion)
- Hypermetabolic states (e.g., thyrotoxicosis)
- Massive resuscitation with hyperchloremic fluids
- Hyperthermia
- Hypothermia
- Severe diarrhea
- Diabetic ketoacidosis

Prevention

No specific prevention except for treatment of the underlying condition.

Special Considerations

- Monitor patients with acid base disorders closely and adjust treatment for a change in clinical status (e.g., adjust minute ventilation as metabolic acidosis improves to prevent rebound alkalosis).
- Do not reduce PaCO₂ to <25 mm Hg, as this may produce hypocarbia-induced vasoconstriction and ischemia.
- Resuscitation with normal saline in shock states can worsen metabolic acidosis.
- Respiratory acidosis can be corrected by decreasing PaCO₂. Metabolic acidosis can be buffered acutely by inducing a respiratory alkalosis.

Further Reading

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Acute Adrenal Insufficiency

Definition

Failure of the hypothalamic-pituitary adrenal axis to produce sufficient glucocorticoids and/or mineralocorticoids.

Presentation

- Nausea, vomiting
- Fever
- Acute dehydration
- Tachycardia
- Hypotension/shock, often refractory to fluids and/or vasopressors
- Hyponatremia
- Hypokalemia
- Hypoglycemia

Pathophysiology

Corticotrophin-releasing hormone (CRH) is released from the hypothalamus and stimulates the pituitary to release

adrenocorticotrophic hormone (ACTH). Adrenocorticotrophic hormone then causes the adrenal glands to release cortisol. Cortisol provides negative feedback, inhibiting the release of CRH and ACTH. Adrenal insufficiency (AI) can be caused by destruction of the adrenal cortex, pituitary disease, or exogenous steroids. Primary AI is caused by destruction of the cortex, most commonly due to autoimmune reaction, hemorrhage, or infection. It is associated with both cortisol and mineralocorticoid deficiency. Secondary AI is caused by pituitary lesions; tertiary AI is caused by dysfunction of the hypothalamus. Both are associated only with cortisol deficiency. Suppression of adrenal function by exogenous glucocorticoids administration can also cause AI. *Relative or functional AI* is defined as a decreased level of glucocorticoids that may be adequate under normal situations but is insufficient under physiologic stress.

DIFFERENTIAL DIAGNOSIS

- Septic shock
- Acute abdomen

129

Immediate Management

- Administer steroids (hydrocortisone 100 mg IV or dexamethasone 4 mg IV). Hydrocortisone is preferred in primary AI with hyperkalemia because of its mineralocorticoid activity, but dexamethasone may be used because it does not affect serum cortisol assays.
- Restore intravascular volume with isotonic (normal saline or LR) fluids. Hypotonic saline may worsen hyponatremia.
- Correct hypoglycemia with a dextrose infusion (25 g dextrose in 50 mL of water).
- Support blood pressure with vasopressors. (Consider a norepinephrine infusion, but hypotension in AI may be resistant to catecholamines. Vasopressin infusion is an alternative.)

Diagnostic Studies

- Plasma electrolytes and glucose
- Baseline cortisol, renin, and ACTH levels. (If possible, draw labs before steroid administration but do not delay therapy for blood draws.)
- Consider an ACTH stimulation test.

Subsequent Management

- Evaluate the patient for the precipitating cause of AI.

- Stress dose steroids can be tapered to maintenance doses over the course of several days depending on the course of the underlying illness.
- Although the initial doses of steroids have sufficient mineralocorticoid activity, mineralocorticoid replacement may be eventually required in patients with primary adrenal insufficiency in order to prevent sodium loss and associated volume depletion and hyperkalemia.
- Consider consulting an endocrinologist.

Risk Factors

- Known chronic adrenal insufficiency
- Corticosteroid use (more than prednisone 20 mg/day or equivalent) for >4 weeks within the preceding year
- Abrupt discontinuation of exogenous corticosteroids.
- Severe sepsis

130

Prevention

A careful history and physical examination may elicit steroid use or signs of chronic AI. Patients at risk should receive perioperative stress dose steroids. For major surgical stress, administer hydrocortisone 100 mg before induction of anesthesia followed by 50 mg every 8 hours for 24 hours and then taper over several days to baseline dose.

Special Considerations

- Patients with septic shock may develop relative adrenal insufficiency without a previous history of steroid use or other risk factors. Adrenal insufficiency should be suspected in patients with hypotension that is unresponsive to vasopressor or fluid therapy. Although the data are conflicting, low-dose steroids (200–300 mg/day hydrocortisone) appear to reduce the time to reversal of septic shock but have no clear benefit on overall mortality.

Further Reading

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Charmandari E, Nicolaides NC, Chrousos GP. Adrenal insufficiency. *Lancet.* 2014; 383(9935): 2152–2167.

Alkalosis

Definition

Blood pH >7.45 (normal 7.36–7.45) caused by either hypocarbia, metabolic effects (i.e., increased plasma HCO_3), or both.

Presentation

- Mild alkalosis is often asymptomatic.
- Laboratory results on arterial or venous blood gas analysis
- Respiratory alkalosis symptoms include paresthesias, carpopedal spasm, and lightheadedness.
- Metabolic alkalosis symptoms are usually related to the underlying etiology (e.g., hypovolemia) and/or the associated electrolyte abnormalities (e.g., hypokalemia).

Pathophysiology

Alkalosis is caused by loss of acid from the extracellular space (e.g., hypochloremia), excessive HCO_3 loads (e.g., administration of sodium bicarbonate to treat lactic acidosis) or hyperventilation that acutely reduces PaCO_2 . Compensation may be prevented by inability to excrete the excess HCO_3 in urine due to intravascular volume depletion (caused by hypovolemia or factors such as heart failure or cirrhosis), renal insufficiency, chloride depletion, or hypokalemia.

DIFFERENTIAL DIAGNOSIS

- Laboratory error

Immediate Management

- Establish IV access.
- Mild cases of alkalosis do not necessarily need urgent treatment.
- If the patient is mechanically ventilated, adjust minute ventilation to increase PaCO_2 while ensuring adequate oxygenation (10 mm Hg increase in PaCO_2 produces approximately 0.08 decrease in pH).
- Correct the effective volume deficit for true volume deficit, replete with isotonic or hypertonic saline.
- In patients who are edematous (e.g., cirrhosis, heart failure), alkalosis may be caused by diuretic induced hypokalemia. If this is suspected, administer potassium chloride.
- Administer potassium chloride for patients with potassium and chloride depletion.

Diagnostic Studies

- Basic metabolic panel
- Arterial blood gas analysis
- Urinary chloride

Subsequent Management

- Diagnose and treat the underlying cause.
- Potassium sparing diuretics (e.g., spironolactone 25–200 mg/day) may be helpful in patients with metabolic alkalosis and edematous states that require further diuresis.
- Acetazolamide is a carbonic anhydrase inhibitor and can increase urinary bicarbonate excretion; the effect may take several hours.
- Renal replacement therapy may infrequently be indicated in patients with kidney failure and severe metabolic alkalosis.
- Patients with severe metabolic alkalosis ($\text{pH} > 7.55$) who cannot be dialyzed may rarely be treated with hydrochloric acid infusion (1 liter of 0.1 N solution of HCL over 12–24 hours).

Risk Factors

- Second most common acid-base disorder in hospitalized adults
- Loop diuretic administration
- Severe hypoproteinemia
- Hypocarbica
- Volume contraction
- Hypochloremia
 - Vomiting
 - Proximal enteric fistula
- Iatrogenic causes
 - Hyperventilation
 - Administration of weak ions (acetate, citrate). This occurs most commonly in patients who receive total parenteral nutrition.

Prevention

Avoid inadvertent hyperventilation in mechanically ventilated patients. Monitor acid-base status in patients who are receiving diuretics.

Special Considerations

- Regardless of the specific etiology, alkalosis generally responds to either PaCO_2 regulation or chloride administration.

Further Reading

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Anaphylaxis

Definition

An acute, severe allergic or hypersensitivity reaction that is caused by release of inflammatory mediators and cytokines from mast cells and basophils.

Presentation

- Respiratory compromise (dyspnea, bronchospasm, hypoxemia)
- Hypotension
- Tachycardia
- Urticaria
- Angioedema
- Gastrointestinal symptoms (nausea, vomiting, diarrhea, crampy abdominal pain)

Pathophysiology

Massive release of leukotrienes, histamine, and prostaglandins in response to release of IgE from mast cells triggered by an allergen such as food, medication, or insect bite. Anaphylaxis can be associated with potentially life-threatening vasodilation, myocardial suppression, bronchoconstriction, and tissue edema.

DIFFERENTIAL DIAGNOSIS

- Shock (e.g., sepsis, hypovolemia, cardiogenic shock)
- Nonimmunologic drug reaction
 - Red man syndrome caused by rapid administration of vancomycin
 - Morphine-induced histamine release
- Drug overdose
- Malignant hyperthermia
- Acute asthma exacerbation
- Nonallergic angioedema
- Acute generalized urticaria

Immediate Management

- Evaluate the patient's airway and consider endotracheal intubation
- Administer supplemental O₂ as needed to treat hypoxia
- Establish large-bore IV access
- Treat bronchospasm (inhaled albuterol 4–8 metered doses)
- Anaphylaxis without pulmonary/cardiovascular compromise
 - Administer hydrocortisone 100 mg IV
 - Administer diphenhydramine 50 mg IV (H₁ antagonist).
Note: This will not relieve airway obstruction, hypotension, or shock.
- Anaphylaxis with cardiovascular compromise
 - Preceding interventions **plus**
 - Consider intubation and mechanical ventilation
 - Arterial blood gas to evaluate acid-base status, hypoxia, hypoventilation
 - Administer isotonic crystalloid or colloid solutions to replace intravascular volume
 - Epinephrine—Titrate to symptom severity and clinical response. No absolute contraindications to its use in anaphylaxis.
 - Epinephrine 0.15–0.3 mg IM (auto-injector)
 - Epinephrine 1 mg IV bolus for cardiac arrest
 - Epinephrine infusion (start at 2–10 mcg/min, titrate to effect)
 - For anaphylaxis resistant to epinephrine, use
 - Norepinephrine infusion (initial dose 0.05–0.1 mcg/kg/min)
 - Glucagon for patients on beta-blockers (initial dose 1–5 mg over 5 minutes followed by infusion 5–15 mcg/min)
 - Vasopressin (0.04 U/min)
- Search for and discontinue the triggering agent. Perform a careful evaluation of all agents administered immediately prior to the anaphylactic event, including antibiotics, latex, and neuromuscular blocking agents.
- Inform the surgical team and consider terminating the procedure if possible.

Diagnostic Studies

- Predominantly a clinical diagnosis.
- Histamine levels peak almost immediately after the reaction and if measured should be drawn within an hour of the reaction.

- A tryptase level can be drawn 15 minutes to 3 hours after the onset of symptoms. Note: A normal level does not exclude an allergic reaction.

Subsequent Management

- Document the event and the trigger to avoid future exposure.
- If the trigger is unknown, consider further evaluation by an allergy specialist for skin testing and/or measurement of specific IgE.

Risk Factors

- Prior history of allergic reactions
- Allergic rhinitis
- Asthma

Prevention

- Review carefully each patient's record for known allergies and previous reactions.
- Limit availability and exposure to latex products.

Special Considerations

- Although anaphylaxis may occur at any time during the operation, most reactions occur around the time of induction.
- Neuromuscular agents are the most common trigger in the perioperative period followed by latex and antibiotics. Local anesthetics have been implicated in anaphylactic reactions, but local anesthetic toxicity should be ruled out first. Any agent or medication may cause anaphylaxis.

Further Reading

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Diabetes Insipidus

Definition

Disorder of water metabolism leading to polyuria (>3 L/day in adults) due to either deficient secretion of antidiuretic hormone (ADH) (central diabetes insipidus [DI]) or renal resistance to ADH (nephrogenic DI).

Presentation

- Polyuria (>200 mL/h)
- Nocturia
- Polydipsia
- Hypernatremia
- Low urine osmolality (<150 mOsm/kg)
- Hypovolemia
- Hypotension

Pathophysiology

Antidiuretic hormone, also known as vasopressin, increases water absorption in the kidney collecting ducts, concentrating urine and decreasing its volume. A precursor of ADH is synthesized in the hypothalamus. Antidiuretic hormone is stored and released from the posterior pituitary in response to reduced plasma volume. Central or neurogenic DI results from lack of ADH secondary to injury to the posterior pituitary gland, pituitary stalk, or the anterior hypothalamus. Most common causes include pituitary surgery, traumatic brain injury, intracranial hypertension, brain death, tumors, and infections. Nephrogenic DI in adults is typically associated with chronic lithium use or hypercalcemia.

DIFFERENTIAL DIAGNOSIS

- Psychogenic polydipsia
- Osmotic (including hyperglycemia) diuresis
- Diuretic use
- Fluid overload
- Fluid mobilization

Immediate Management

- Establish adequate IV access
- Correct the free water deficit [deficit (L) = weight (kg) \times 0.6 \times (Na-140)/Na].
- Control polyuria with vasopressin or desmopressin acetate (DDAVP). DDAVP can be given intranasally 10–20 mcg and repeated every 30–60 minutes until urine output is <100 mL/hour. If intranasal route is not feasible, administer DDAVP 2 mcg IV over 2 minutes every 12 hours.

Diagnostic Studies

- Plasma sodium
- Plasma osmolality
- Urine osmolality
- Urine specific gravity

Subsequent Management

- Continue replacing free water deficit either with enteral water or dextrose 5% in water as guided by laboratory studies.
- Replace urine losses hourly.
- Monitor electrolytes and glucose every 4 to 6 hours.
- Redose DDAVP when urine output increases to above 200 mL/hour.
- If patient condition permits, allow unrestricted access to free water.

Risk Factors

- Traumatic brain injury
- Pituitary surgery
- Chronic lithium use
- Brain death

Prevention

Monitor urine output in patients at risk for developing diabetes insipidus.

Special Considerations

- Nephrogenic DI is less commonly seen in acute settings. It is treated with low sodium and low protein diet, thiazide diuretics, and nonsteroidal anti-inflammatory drugs (NSAIDs) that inhibit prostaglandin synthesis.
- Patients with free access to water may be able to drink sufficient amounts to compensate for urinary loss.

Further Reading

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Diabetic Ketoacidosis and Hyperosmolar Hyperglycemia

Definition

Diabetic ketoacidosis (DKA) is characterized by hyperglycemia, anion gap metabolic acidosis, ketosis, and severe volume deficit. Serum glucose is typically >500 mg/dL. In hyperosmolar hyperglycemia (HHS) there is no (or very little) ketoacidosis, elevation of

serum osmolality, and serum glucose is frequently >1000 mg/dL. These are two of the most serious acute complications of diabetes.

Presentation

- DKA usually evolves rapidly over a 24-hour period. Hyperosmolar hyperglycemia typically has a slower onset that may take several days.
- Polyuria
- Polydipsia
- Weight loss
- Neurologic symptoms (lethargy, obtundation, coma)
- Hyperventilation (DKA)
- Abdominal pain (DKA)
- Hyperglycemia
- Acidosis (DKA)

Pathophysiology

Diabetic ketoacidosis and hyperosmolar hyperglycemia are caused by either a relative or absolute lack of insulin in combination with a precipitating factor (most commonly an infection). Fluid, electrolyte, and acid-base balance are all affected. Hyperglycemia causes osmotic diuresis with subsequent fluid and electrolyte deficits. In DKA, unopposed glucagon leads to increased lipolysis and the formation of ketones. Severe acidosis increases the required minute ventilation for pH buffering.

DIFFERENTIAL DIAGNOSIS

- Lactic acidosis
- Renal failure
- Alcoholic ketoacidosis
- Fasting ketoacidosis
- Sepsis
- Aspirin overdose
- Other conditions that can cause metabolic acidosis

Immediate Management

- Assess airway, breathing, circulation, and mental status.
- Establish IV access.
- Assess fluid status.
 - Hypotensive patients should be given isotonic fluids (0.9% NaCl) as quickly as possible to restore volume and blood pressure.

Immediate Management (continued)

- Normotensive patients with mild volume deficits can be given fluids at 2–3 times the maintenance rate (250–500 mL/h). Use 0.9% saline in patients with low serum sodium and 0.45% saline for high or normal serum sodium.
- Check electrolytes hourly.
- Assess acid-base status
 - If pH is <7.0, consider bicarbonate (100 mmol in 400 mL H₂O with 20 mEq KCl over 2 hours) especially in patients with decreased cardiac contractility and vasodilation.
- Insulin deficiency
 - Start continuous regular insulin infusion (suggested initial dose 0.1 U/kg bolus followed by a 0.1 U/kg/h infusion).
 - Double the dose if serum glucose does not fall 50–70 mg/dL in the first hour.
- Assess electrolytes
 - Replete serum potassium to 3.3 mEq/L

139

Diagnostic Studies

- Electrolytes and glucose
 - Plasma osmolality
 - Urine and serum ketones
 - Arterial blood gas
 - Complete blood count
 - Additional workup directed to possible inciting etiologies

Subsequent Management

- Add 5% dextrose to the intravenous fluids when serum glucose reaches 200 mg/dL.
- Decrease the insulin infusion and begin transition to subcutaneous insulin sliding scale once glucose is <200 mg/dL.
- Postpone elective surgery and if feasible delay urgent surgery while the patient is resuscitated and electrolytes, pH, and perfusion normalize.
- Identify and treat the inciting cause.

Risk Factors

- Diabetes mellitus
- Discontinuation of insulin in a diabetic patient
- Acute illness or emergency surgery in a diabetic patient

Prevention

Early detection and treatment of hyperglycemia and diabetes mellitus.

Special Considerations

- Treatment of acidosis in DKA with bicarbonate remains controversial.
- Avoid subcutaneous insulin administration in DKA and HHS due to variable absorption.
- Hyperglycemia and dehydration may induce hypernatremia that is often artifactual and not clinically significant.
- K^+ and Mg^{2+} are cotransported with glucose and should be simultaneously repleted to prevent life-threatening dysrhythmias.

Further Reading

Gouveia CF, Chowdhury TA. Managing hyperglycaemic emergencies: an illustrative case and review of recent British guidelines. *Clin Med*. 2013; 13(2): 160–162.

Russo N. Perioperative glycemic control. *Anesthesiol Clin*. 2012; 30(3): 445–466.

Sebranek JJ, Lugli AK, Coursin DB. Glycaemic control in the perioperative period. *Br J Anaesth*. 2013; 111(Suppl 1): 18–34.

Hypercalcemia

Definition

Calcium (Ca^{2+}) level >10.5 mg/dL (normal, 8.5–10.5 mg/dL)

Presentation

- Incidental laboratory finding
- Mental status changes
- Hyperreflexia
- Hypertension
- Bradycardia
- Cardiac arrest
- Nausea/vomiting
- Polyuria
- Renal calculi
- Oliguric renal failure

Pathophysiology

The most common causes of hypercalcemia are metastatic disease, paraneoplastic syndrome, secondary hyperparathyroidism, lithium or thiazide toxicity, and immobility.

DIFFERENTIAL DIAGNOSIS

- Laboratory error

Immediate Management

- Evaluate airway, breathing, and circulation.
- Administer 0.9% normal saline solution.
- Administer furosemide 40 mg IV in patients with normal renal function in order to increase urinary calcium excretion.
- Begin hemodialysis in patients with renal failure.
- Discontinue pharmacologic agents associated with hypercalcemia (e.g., thiazides, calcium carbonate, lithium, theophylline).

Diagnostic Studies

- Basic metabolic panel including Ca^{2+} , Mg^{2+} , and phosphorus levels
- Liver function tests
- Parathyroid hormone (PTH) level
- Amylase level
- 12-Lead electrocardiogram

Subsequent Management

- Bisphosphonate therapy
- Calcimimetic agents (e.g., cinacalcet)
- Calcitonin therapy
- Investigate underlying cause if unknown.
- Workup for malignancy
- Follow amylase levels until Ca^{2+} returns to normal.

Risk Factors

- Malignancy (especially breast, lung, kidney, multiple myeloma, lymphoma)
- Hyperparathyroidism
- Kidney failure
- Granulomatous diseases: tuberculosis, sarcoidosis

Prevention

Maintain adequate hydration and urine output with sodium-containing fluids in patients at risk for hypercalcemia. Avoid prolonged bed rest in patients.

Special Considerations

- Avoid salt restriction and thiazide diuretics in patients at risk for hypercalcemia.
- Consider using reduced doses of neuromuscular blocking agents in patients with muscle weakness.
- Hypercalcemia can cause pancreatitis.

Further Reading

Chang WT, Radin B, McCurdy MT. Calcium, magnesium, and phosphate abnormalities in the emergency department. *Emerg Med Clin North Am.* 2014; 32(2): 349–366.

Kaye AD, Riopelle JM. Intravascular fluid and electrolyte physiology. In: Miller RD, ed. *Miller's anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1711–1712.

Hypocalcemia

Definition

- Calcium (Ca^{2+}) level <85 mg/dL (normal, 8.5–10.5 mg/dL)
- Ionized calcium level <2.0 meq/L (normal, 2.0–2.5 meq/L)

Presentation

- Incidental laboratory finding
- Mental status changes
- Tetany
- Chvostek sign (momentary contraction of the facial muscles in response to tapping the facial nerve)
- Trousseau sign (flexion of the wrist and metacarpophalangeal joints, extension of the DIP and PIP joints, and finger adduction in response to an inflated blood pressure cuff)
- Perioral numbness or tingling
- Laryngospasm
- Hypotension
- Prolongation of Q–T interval or heart block
- Seizures

Pathophysiology

The most common cause of low total serum calcium is hypalbuminemia secondary to cirrhosis, nephrosis, malnutrition, burns,

chronic illness, or sepsis. This may not accurately represent the physiologically important ionized calcium concentration. Other causes include hypoparathyroidism, pseudohypoparathyroidism, hypomagnesemia, vitamin D deficiency, and chronic kidney disease.

DIFFERENTIAL DIAGNOSIS

- Laboratory error

Immediate Management

- Administer CaCl_2 1 g IV over 5 minutes
- Repeat as needed to return ionized Ca^{2+} levels to normal or until symptom resolution.

Diagnostic Studies

- Basic metabolic panel including Ca^{2+} , Mg^{2+} , phosphorus levels
- Ionized calcium
- If ionized calcium level is not available, calculate the corrected total calcium:

Corrected $\text{Ca}^{2+} = (\text{measured total } \text{Ca}^{2+}) + 0.8 * (4.1 - \text{patient's albumin})$

- Liver function tests, including albumin
- Parathyroid hormone level
- 12-Lead ECG

Subsequent Management

- Investigate the cause of hypocalcemia if unknown.
- Maintain normocalcemia with calcium gluconate via IV bolus or continuous infusion (less irritating to veins).

Risk Factors

- Low albumin level
- Acute renal failure or chronic kidney disease
- Rapid transfusion of citrated blood products (1.5 mL/kg/min)
- Acute hyperventilation
- Hyperparathyroidism
- Sepsis
- Thyroidectomy or parathyroidectomy
- Acute panhypopituitarism
- Hypomagnesemia

Prevention

- Consider administration of 500 mg CaCl_2 for every 8–10 units of packed red blood cells (PRBC) during rapid transfusion.

- Alternatively, measure ionized Ca^{2+} every 5 units and treat as necessary.

Special Considerations

- CaCl_2 provides immediately available calcium. Calcium gluconate requires degluconation by the liver before the calcium is biologically available. Hepatic dysfunction may limit the timely availability of calcium gluconate.
- Hypocalcemia may be accompanied by hypomagnesemia in the setting of large-volume resuscitation with isotonic saline.

Further Reading

Chang WT, Radin B, McCurdy MT. Calcium, magnesium, and phosphate abnormalities in the emergency department. *Emerg Med Clin North Am.* 2014; 32(2): 349–366.

Kaye AD, Riopelle JM. Intravascular fluid and electrolyte physiology. In: Miller RD, ed. *Miller's anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1711–1712.

Hyperkalemia

Definition

Elevated serum potassium (normal typically 3.5–5.0 mEq/L).

Presentation

- Incidental laboratory finding
- Nausea, vomiting
- Muscle weakness, paresthesias, paralysis
- Cardiac conduction abnormalities and cardiac arrhythmias. Peaked T waves with shortened QT interval are typically the first findings. In more severe cases, there is progressive PR interval and QRS prolongation leading to a sine wave pattern on ECG and cardiac arrest. Electrocardiogram changes do not always correlate with the serum potassium concentration and also depend on the acuteness of the potassium elevation.

Pathophysiology

High plasma concentration of potassium impairs myocardial conduction and may potentially result in cardiac arrest.

DIFFERENTIAL DIAGNOSIS

- Laboratory error
- Improper sample handling or mechanical trauma during venipuncture (cell lysis)

- Severe leukocytosis or thrombocytosis can cause artificial potassium elevation

Immediate Management

- Establish IV access.
- Obtain a 12-lead ECG and establish continuous ECG monitoring.
- If ECG changes are significant, administer 500–1000 mg calcium chloride to stabilize cardiac membranes. Can be repeated after 5 minutes if ECG changes persist.
- Administer 10 units of regular insulin IV along with 50 mL of D₅₀W IV to temporarily shift potassium into cells.

Diagnostic Studies

- Plasma electrolytes, blood urea nitrogen (BUN), creatinine
- Electrocardiogram

Subsequent Management

- Treat the underlying cause(s).
- Reduce excess potassium from the body.
 - Loop and thiazide diuretics combined with normal saline hydration increase potassium loss in urine.
 - Administration of a cation exchange resin (e.g., sodium polystyrene sulfonate **without sorbitol** [15–30 g orally every 4–6 hours]) can lower serum potassium but is not effective in the acute phase and should be used with extreme caution, especially in patients with suspected ileus or bowel obstruction.
 - Hemodialysis, especially in patients with severe hyperkalemia, renal failure, or severe tissue breakdown (e.g., crush injury)

Risk Factors

- Acute and chronic renal failure
- Metabolic acidosis
- Insulin deficiency (diabetic ketoacidosis, hyperosmolality)
- Increased tissue catabolism
- Tissue or cell destruction: tissue necrosis, hemolysis, skeletal muscle crush injury
- Strenuous exercise
- Use of depolarizing neuromuscular blockers in patients with severe burn injury or upper motor neuron disorders (e.g., cerebral pathology, spinal cord injury)
- Iatrogenic (massive transfusion, inadvertent overdose)

Prevention

Avoid iatrogenic causes. Monitor potassium levels closely in patients with risk factors.

Special Considerations

- Therapy for hyperkalemia addresses three major aims: (1) supporting myocardial polarization and depolarization; (2) relocating potassium from the plasma space to the intracellular space; and (3) reducing the total body potassium load.

Further Reading

Kaye AD, Riopelle JM. Intravascular fluid and electrolyte physiology. In: Miller RD, ed. *Miller's anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1710.

Hypokalemia

146

Definition

Low serum potassium (normal 3.5–5.0 mEq/L, mild 3.0–3.5 mEq/L, moderate 2.5–3.0 mEq/L, and severe <2.5 mEq/L).

Presentation

- Incidental laboratory finding
- Severe muscle weakness. Typically begins in lower extremities and progresses to trunk and the upper extremities.
- Respiratory failure due to respiratory muscle weakness
- Rhabdomyolysis
- Cardiac arrhythmias—premature atrial and ventricular beats, bradycardia, paroxysmal atrial or junction tachycardias, atrioventricular block, ventricular tachycardia, or fibrillation.
- Electrocardiogram abnormalities—ST segment depression, inverted T waves and U waves.

Pathophysiology

Most cases of hypokalemia results from loss of potassium from gastrointestinal or urinary tracks that are not appropriately repleted. The severity of symptoms is dependent on the degree and duration of the hypokalemia.

DIFFERENTIAL DIAGNOSIS

- Laboratory error
- Hypomagnesemia

- Hypocalcemia
- Cushing syndrome

Immediate Management

- Establish IV access.
- Initiate ECG monitoring.
- Slowly administer potassium chloride 20 mEq IV for life-threatening hypokalemia.
- Limit infusion to 20 mEq/hour except in the setting of life-threatening dysrhythmias with careful ECG monitoring.

Diagnostic Studies

- Plasma electrolytes, including magnesium, BUN, creatinine
- 12-Lead ECG

Subsequent Management

- Consider administration through a central venous catheter because potassium chloride infusions are extremely irritating to peripheral veins.
- Treat or reverse underlying etiology of potassium loss (e.g., hold potassium-wasting diuretics, gastrointestinal [GI] losses).
- Correct coexisting hypomagnesemia.
- If appropriate, additional oral potassium replacement can be administered.
- Continue telemetry monitoring postoperatively.

Risk Factors

- Diuretic administration
- Large volume resuscitation
- Alkalosis (including respiratory alkalosis due to hyperventilation)
- Gastrointestinal losses (diarrhea, vomiting, ileal conduit)
- Uncontrolled diabetes (DKA, HHS)
- Salt-wasting nephropathies
- Primary aldosteronism
- Beta-agonist use

Prevention

Monitor plasma electrolytes in patients at risk for hypokalemia.

Special Considerations

- Mild or moderate hypokalemia rarely requires emergency treatment.

- Potassium is principally an intracellular cation. Low plasma levels imply that the intracellular stores are depleted. Severe hypokalemia may require as much as 200–300 mEq of potassium to restore normal plasma and intracellular levels and may require several days of intravenous therapy to correct.
- In DKA and HHS, the insulin deficiency favors movement of potassium out of cells. The patient may therefore have a total body potassium deficit despite a normal or slightly low serum potassium level. Insulin therapy to treat the hyperglycemia may worsen hypokalemia.

Further Reading

Kaye AD, Riopelle JM. Intravascular fluid and electrolyte physiology. In: Miller RD, ed. *Miller's anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1710.

Hypermagnesemia

148

Definition

Magnesium (Mg^{2+}) level >2.5 mEq/L (normal 1.5–2.0 mEq/L).

Presentation

- Incidental laboratory finding
- Neuromuscular effects (in order of increasing Mg levels)
 - Diminished deep tendon reflexes (first sign)
 - Headache, lethargy, drowsiness
 - Muscle weakness
 - Somnolence, loss of deep tendon reflexes, muscle paralysis
 - Flaccid quadriplegia
 - Fixed and dilated pupils mimicking brain stem herniation
- Cardiovascular (in order of increasing Mg levels)
 - Bradycardia, hypotension
 - PR, QRS prolongation
 - Increase in Q-T interval
 - Complete heart block
 - Cardiac arrest
- Hypocalcemia

Pathophysiology

Most commonly caused by excessive intake of Mg^{2+} (especially laxatives) or in patients with renal impairment. High magnesium levels decrease impulse transmission across the neuromuscular junction.

DIFFERENTIAL DIAGNOSIS

- Laboratory error

Immediate Management

- Assess airway, ventilation, and mental status. Intubate the trachea and initiate mechanical ventilation as necessary.
- Control the underlying cause (e.g., stop the infusion).
- Administer calcium chloride 1 g IV over 5 minutes in severe hypermagnesemia.
- Intravenous isotonic fluid therapy (normal saline or lactated Ringer's) plus loop diuretics can increase renal excretion of magnesium.

Diagnostic Studies

- Electrolytes including magnesium, calcium, and phosphate
- BUN, creatinine
- 12-Lead ECG

Subsequent Management

- Cessation of magnesium therapy should be enough to reduce the magnesium level in patients with normal renal function.
- Hemodialysis may be necessary in patients with severe symptoms and acute or chronic renal failure.

Risk Factors

- Aggressive treatment of pre-eclampsia
- Use of Mg^{2+} containing antacids or laxatives
- Renal failure

Prevention

Monitor Mg levels when using it therapeutically especially in patients with renal insufficiency.

Special Considerations

- Magnesium and calcium antagonize each other's effects.
- Mg^{2+} will produce profound muscle weakness in patients with myasthenia gravis or Lambert-Eaton syndrome.
- Mg^{2+} prolongs the action of neuromuscular blocking agents.

Further Reading

Herroeder S, Schonherr M et al. Magnesium—essentials for anesthesiologists. *Anesthesiology*. 2011; 114(4): 971–993.

Kaye AD, Riopelle JM. Intravascular fluid and electrolyte physiology. In: Miller RD, ed. *Miller's anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1713–1714.

Hypomagnesemia

Definition

Magnesium (Mg^{2+}) level <1.5 mEq/L (normal 1.5–2.0 mEq/L).

Presentation

- Incidental laboratory finding
- Neuromuscular: tremor, involuntary movements, tetany, weakness, altered mental status, seizures, delirium, coma
- Cardiovascular: QRS widening, peak T waves with moderate hypomagnesemia. More severe deficits can lead to widening of PR interval, decreased T waves, and atrial and ventricular arrhythmias including torsades des pointes.
- Hypocalcemia, hypoparathyroidism, PTH resistance
- Hypokalemia

Pathophysiology

Most commonly occurs due to gastrointestinal (diarrhea) or renal losses. It is also associated with hypokalemia and hypocalcemia.

DIFFERENTIAL DIAGNOSIS

- Laboratory error

Immediate Management

- Establish IV access and ECG monitoring.
- In hemodynamically unstable patients, given 1–2 g of magnesium sulfate over 2–15 minutes.
- In hemodynamically stable patients, the amount of replacement can be estimated from the plasma level:

Mg level	Replacement dose (IV)
2.0–2.25	2.0 g
1.75–1.9	4.0 g
1.5–1.74	6.0 g
1.25–1.49	8.0 g
1.0–1.24	10.0 g

Diagnostic Studies

- Electrolytes including magnesium, calcium, and phosphate
- Blood urea nitrogen, creatinine
- 12-Lead ECG

Subsequent Management

- Serial serum Mg levels
- Patients with minimal or no symptoms can receive oral replacement. This may cause diarrhea and other GI symptoms.

Risk Factors

- Chronic diarrhea
- Proton pump inhibitor therapy
- Alcohol abuse
- Diuretic use
- Large volume fluid resuscitation
- Very common in critically ill patients

Prevention

Careful monitoring of magnesium levels in high-risk patients.

Special Considerations

- Hypomagnesemia is associated with an increased risk of perioperative arrhythmias and bronchospasm.
- Serum Mg level should be monitored during administration in patients with acute or chronic renal insufficiency.

Further Reading

Herroeder S, Schonherr M, et al. Magnesium—essentials for anesthesiologists. *Anesthesiology*. 2011; 114(4): 971–993.

Kaye AD, Riopelle JM. Intravascular fluid and electrolyte physiology. In: Miller RD, ed. *Miller's anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1713–1714.

Hypernatremia

Definition

Sodium level >145 mEq/L (normal sodium, 135–145 mEq/L).

Presentation

- Incidental laboratory finding
- Severe dehydration (hypotension, oliguria, decreased skin turgor)
- Mental status changes
- Seizures

Pathophysiology

Excessive free water loss is the most common cause. Excessive sodium administration may also result in hypernatremia.

DIFFERENTIAL DIAGNOSIS

- Laboratory error

Immediate Management

- Hypovolemic hypernatremia:
 - Administer fluids to correct hypovolemia
 - Hypotonic fluid: 0.45% saline or D₅W (free water) to correct Na⁺
- Normovolemic hypernatremia:
 - Hypotonic fluid: 0.45% saline or D₅W to correct Na⁺
 - Correct underlying cause (e.g., administer DDAVP for diabetes insipidus)
- Hypervolemic hypernatremia:
 - Discontinue Na⁺ containing solutions
 - Consider furosemide (20 mg IV as a starting dose) if appropriate for the patient's renal function

Diagnostic Studies

- Basic metabolic panel, including Ca²⁺, Mg²⁺, and phosphorus
- Serum osmolality
- Urine electrolytes and creatinine
- Urine osmolality
- 24-Hour urine volume

Subsequent Management

- Monitor fluid and electrolyte intake carefully.
- Monitor urine output carefully.
- Follow serial sodium trends in order to control the rate of sodium correction.
- Determine the cause of hypernatremia and treat appropriately.

Risk Factors

- Advanced age
- Hospitalization: tube feeding, mechanical ventilation, hypertonic infusions
- Hypovolemic patients: open wounds, GI losses, insufficient ACTH, mannitol or lactulose administration, loop diuretic use in conjunction with a salt-restricted diet

Risk Factors (continued)

- Euvolemic patients: diabetes insipidus (nephrogenic or central), lithium toxicity
- Hypervolemic patients: iatrogenic (NaHCO_3 or hypertonic saline administration)

Prevention

Follow plasma electrolyte levels closely in patients receiving large quantities of sodium-containing fluids, as well as those with large-volume losses from diuretics, lactulose, or other sources.

Special Considerations

- Acute onset is <24 hours; chronic onset is >24 hours.
- In acute hypernatremia, correct the serum sodium at a rate of 2–3 mEq/L/h (maximum 12 mEq/L/day).
- For chronic hypernatremia, correct the serum sodium at a rate not exceeding 0.5 mEq/L/h.
- Free water deficit may be calculated using the following equation:

$$\text{Free H}_2\text{O deficit} = \text{Body water (L/kg)} \times \text{weight (kg)} \times ((\text{serum Na}^+ / 140) - 1)$$

- Estimate body water as 0.6 L/kg in males and 0.5 L/kg in females.

Further Reading

Bagshaw SM, Townsend DR, McDermid RC. Disorders of sodium and water balance in hospitalized patients. *Can J Anaesth.* 2009; 56(2): 151–167.

Kaye AD, Riopelle JM. Intravascular fluid and electrolyte physiology. In: Miller RD, ed. *Miller's anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1706–1709.

Lindner G, Funk GC. Hypernatremia in critically ill patients. *J Crit Care.* 2013; 28(2): 216, e11–e20.

Hyponatremia**Definition**

Sodium level <135 mEq/L (normal sodium is 135–145 mEq/L).

Presentation

- Incidental laboratory finding
- Nausea
- Mental status changes
- Seizures
- Cerebral edema leading to tentorial herniation and death

Pathophysiology

Many patients have dilutional hyponatremia and not a true total body Na^+ deficiency. Dilutional hyponatremia is characterized by low Na^+ but nearly normal Cl^- , accompanied by dilute urine and a normal to high urine Na^+ .

Hypovolemic hyponatremia occurs with a decrease in total body water and a greater decrease in total body sodium.

DIFFERENTIAL DIAGNOSIS

- Laboratory error
- Sample dilution (drawing from the same arm as an IV running hyponatremic fluid)
- Hyperglycemia

Immediate Management

- Establish IV access.
- Monitor ECG or hemodynamics as indicated.
- In patients with dilutional hyponatremia:
 - Restrict free water intake
 - Administer furosemide 20–40 mg IV if patient is hypervolemic and renal function is appropriate.
- In hypovolemic patients: Administer 0.9% saline.
- In severe ($\text{Na}^+ < 125 \text{ mEq/L}$) or symptomatic hyponatremia, consider intravenous 3% hypertonic saline
- Calculate sodium deficit using the formula:

$$\text{Sodium deficit (mEq)} = 0.6 \times \text{body weight (kg)} \times (\text{pts Na}^+ - \text{desired Na}^+)$$

Diagnostic Studies

- Basic metabolic panel
- Serum osmolality
- Urine osmolality
- Urine sodium

Subsequent Management

- Acute hyponatremia is <48 hours; chronic hyponatremia is >48 hours.
- For acute hyponatremia, correction of serum sodium should occur at a rate of 1–2 mEq/L/h until serum sodium is 125 mEq/L and symptoms subside.
- In chronic hyponatremia, the rate of serum sodium correction should not exceed 0.5 – 1 mEq/L/h or 12 mEq/L/day.

Caution: Rapid correction may lead to central pontine myelinolysis, an irreversible demyelinating disorder that causes permanent neurologic injury.

Risk Factors

- Excess free water or hypotonic fluid administration.
- Comorbidities including congestive heart failure, liver cirrhosis, and renal failure.
- Absorption of irrigation fluid containing large amounts of free water (during transurethral prostate resection or endoscopic genitourinary or gynecological procedures)
- Thiazide diuretic administration
- Adrenocortical insufficiency
- Hypothyroidism
- Syndrome of inappropriate antidiuretic hormone (SIADH)
- Alcoholism

Prevention

Measure serum electrolyte levels in patients with risk factors.

Special Considerations

- Estimate body water as 0.6 L/kg in males and 0.5 L/kg in females.
- Although ADH antagonist therapy (i.e., aquaretics) is not yet readily available in the United States, these agents will likely replace furosemide, because ADH antagonists induce free water loss with minimal changes in solute excretion.

Further Reading

Bagshaw SM, Townsend DR, McDermid RC. Disorders of sodium and water balance in hospitalized patients. *Can J Anaesth.* 2009; 56(2): 151–167.

Kaye AD, Riopelle JM. Intravascular fluid and electrolyte physiology. In: Miller RD, ed. *Miller's anesthesia*. 7th ed. Philadelphia: Elsevier Churchill Livingstone; 2010:1706–1709.

Hypothermia

Definition

- Body temperature $<35^{\circ}\text{C}$ (normal body temperature is $36.5\text{--}37.5^{\circ}\text{C}$)
- Mild hypothermia: $32\text{--}35^{\circ}\text{C}$

- Moderate hypothermia: 28–32° C
- Severe hypothermia: <28° C

Presentation

- Mild hypothermia:
 - Shivering
 - Lethargy and confusion
 - Weakness
- Moderate hypothermia:
 - Shivering ceases
 - Myocardial depression
 - Dysrhythmias
 - Metabolic acidosis
 - Hyperkalemia
 - Coagulopathy
- Severe hypothermia:
 - Unconsciousness
 - Electrically silent EEG (if <18° C)
 - Ventricular fibrillation or cardiac arrest

Pathophysiology

Hypothermia may be due to increased heat loss from environmental exposure or decreased heat production from thermal dysregulation. During surgery, heat is transferred from the core to the periphery due to anesthesia-induced vasodilation. Heat is also lost during surgery through radiation, evaporation, and convection. Cold-induced diuresis may lead to profound intravascular volume depletion. Impaired thermal regulation (e.g., after injury or drug overdose) results from failure of the hypothalamus to regulate core body temperature. Patients may also present after prolonged exposure to extreme cold (e.g., after a traumatic injury in the winter or after immersion in cold water).

DIFFERENTIAL DIAGNOSIS

- Myxedematous coma (hypothyroidism)

Immediate Management

- Assessment of airway, breathing, circulation.
- Establish an airway and ventilate with 100% O₂.
- If necessary, begin resuscitation according to ACLS guidelines. (Note: A normal cardiac rhythm may not reappear until the patient's core temperature reaches 30° C.)
- Consider administration of Mg²⁺ (2 g of magnesium sulfate IV) for dysrhythmias.

Immediate Management (continued)

- Rewarm the patient:
- Mild to moderate hypothermia:
 - Warm IV fluids
 - Warming blankets
 - Forced-air warmers
- Severe hypothermia:
 - In addition to the preceding:
 - Gastrointestinal lavage via nasogastric tube
 - During surgery, consider body cavity lavage (in particular bladder, and on occasion abdomen or chest) with warm fluid
 - Consider cardiopulmonary bypass support and rewarming (**best option**) in patients with life-threatening hypothermia.

Diagnostic Studies

- Monitor core temperature.
- Thyroid function tests if hypothyroidism is suspected.

Subsequent Management

- **Caution:** Warming-induced peripheral vasodilation in the setting of hypovolemia may lead to significant hypotension requiring hemodynamic support and fluid resuscitation.
- It may be necessary to buffer “washout acidosis” with sodium bicarbonate during rewarming.
- Consider limiting intraoperative time to <2 hours in patients who arrive with or develop hypothermia.

Risk Factors

- Prolonged exposure to cold ambient environment.
- Immersion in cold water.
- Impaired level of consciousness.
- Failure to actively warm the patient during surgery (especially trauma, craniofacial procedures, extensive body cavity surgery)
- Acute alcohol intoxication
- Tranquilizer use (many classes suppress shivering)

Prevention

- Use active warming devices in the operating room (OR) and preoperative holding area.
- Warm the OR.
- Cover the patient to the extent possible.

- Place warming pads on the OR bed.
- Use a forced air warming device during the surgical procedure.
- Use active warming devices for fluids.

Special Considerations

- Patients who are hypothermic on arrival in the OR may be suffering from sepsis, environmental exposure, or injury with acute hemorrhage. Take precautions to prevent further heat loss.
- Shivering, a common sign of core hypothermia is not typically seen in the OR because anesthetics and neuromuscular blocking agents blunt the response to hypothermia.
- Temperature monitoring is recommended as part of the ASA Guidelines on Intraoperative Monitoring.

Further Reading

Horosz B, Malec-Milewska M. Inadvertent intraoperative hypothermia. *Anesthesiol Intensive Ther.* 2013; 45(1): 38–43.

Insler SR, Sessler DI. Perioperative thermoregulation and temperature monitoring. *Anesthesiol Clin.* 2006; 24(4): 823–837.

Malignant Hyperthermia

Definition

Malignant hyperthermia (MH) is a relatively rare inherited disorder of skeletal muscle that causes a hypermetabolic response to a triggering anesthetic agent. Characterized by hyperthermia, body rigidity, and increased CO_2 production, it may be accompanied by cardiovascular collapse or hypertension.

Presentation

- May occur in the operating room or in the early postoperative period
- Increased end-tidal CO_2 production is the first sign
- Tachycardia
- Hypertension
- Muscular rigidity (especially masseter muscle spasm)
- Hyperthermia is a late sign.
- Rhabdomyolysis

Pathophysiology

Malignant hyperthermia is the result of an inborn error of calcium metabolism in skeletal muscle. It is inherited as an autosomal dominant trait.

DIFFERENTIAL DIAGNOSIS

- Light anesthesia (hypertension and tachycardia without CO₂ production)
- Thyrotoxicosis
- Equipment malfunction, increased carbon dioxide, rebreathing, soda lime exhaustion
- Neuroleptic malignant syndrome

Immediate Management

- Call for help and notify the surgeon!
- **Immediately** discontinue triggering anesthetic agent(s): succinylcholine and potent volatile anesthetics. Propofol, narcotics, and N₂O are safe.
- Increase FiO₂ to 100%.
- Hyperventilate the patient with a new anesthesia circuit to treat hypercarbia and respiratory acidosis.
- Establish large-bore IV access.
- Insert an intra-arterial catheter (blood pressure monitoring and frequent blood gas measurements for acid-base status).
- Administer sodium dantrolene (2.5 mg/kg IV until signs and symptoms are controlled or to a maximum of 10 mg/kg).
Note: Dantrolene is difficult and may require several minutes to reconstitute. If possible, one member of the team should be assigned to this task.
- Expand plasma volume with 15 cc/kg bolus × 3 using cool fluids.
- Treat hyperkalemia early.
- Cool the patient aggressively with cold IV solutions. Ask the surgeon to lavage any open body cavity/wound surface with cold irrigating solution. Insert a nasogastric tube and irrigate with ice-cold solution.
- Terminate the surgical procedure as quickly as possible using total intravenous anesthesia.

Diagnostic Studies

- Frequent arterial blood gas and serum electrolyte measurements
- Muscle biopsy for caffeine halothane contracture testing (CHCT) or genetic testing at a separate setting to confirm diagnosis.

Subsequent Management

- Admit patient to the intensive care unit.
- Contact the MHAUS Consultant Hotline (800-644-9737) as soon as possible.

- Maintain urine output at least 2 mL/kg to minimize risk of renal tubular injury from rhabdomyolysis.
- Severe metabolic acidosis may require management with sodium bicarbonate.
- Continue to monitor and correct hyperkalemia.
- Continue dantrolene 1 mg/kg every 4–6 hours for 36 hours after the episode, because the recurrence rate is 25%.
- Discuss the episode with the patient's family and add the patient to the MH national database maintained by the Malignant Hyperthermia Association of the United States (www.mhaus.org).

Risk Factors

- Use of triggering agents (succinylcholine, potent volatile anesthetics) in a patient with a known history of MH.
- Family history, especially an unexplained death of a relative during an anesthetic.
- Malignant hyperthermia may occur despite prior uneventful exposure to triggering agents.

Prevention

Avoid the use of triggering agents in patients with a known history of MH or a suggestive family history.

Special Considerations

- Do not administer calcium channel blockers. In the presence of dantrolene, hyperkalemia and cardiac arrest may result.
- If there is a question as to MH susceptibility, the patient should be managed as if he or she is known to have MH. Because MH is an inherited disorder, consider testing family members for susceptibility.

Further Reading

Benca J, Hogan K. Malignant hyperthermia, coexisting disorders, and enzymopathies: risks and management options. *Anesth Analg*. 2009; 109(4): 1049–1053.

Hopkins PM. Malignant hyperthermia: pharmacology of triggering. *Br J Anaesth*. 2011; 107(1): 48–56.

Myxedema Coma

Definition

An uncommon, life-threatening form of untreated, decompensated hypothyroidism that is precipitated by a secondary insult, such as infection, hypothermia, or medication.

Presentation

- Lethargy, delirium, coma
- Hypothermia
- Bradycardia
- Hypoventilation
- Hyponatremia
- History of generalized fatigue, cold intolerance, constipation, dry skin

Pathophysiology

Physiologic adaptations to long-standing, untreated hypothyroidism include reduced metabolic rate, decreased oxygen consumption, peripheral vasoconstriction, and decreased number of beta-adrenergic receptors. Myxedema coma occurs when these adaptations are not sufficient to compensate for an extreme physiologic stress.

DIFFERENTIAL DIAGNOSIS

- Hypothermia
- Hypoventilation syndrome
- Septic shock

Immediate Management

- Assess airway, breathing, and circulation.
- Intubate the trachea and initiate mechanical ventilation if needed.
- Immediately administer of levothyroxine 500 mcg IV while awaiting results of thyroid function studies, even if diagnosis is only suspected.
- Passively rewarm the patients with blankets and a warm room (rapid warming is contraindicated).
- Pan-culture the patient and start empiric broad spectrum antibiotics.
- Send a cortisol level and then start stress dose steroids while awaiting results.

Diagnostic Studies

- Thyroid function tests including T_3 , T_4 , and TSH levels
- Basic metabolic panel
- Complete blood count with differential
- Arterial blood gas
- Pan-culture
- 12-Lead ECG
- Chest X-ray

Subsequent Management

- Admit to the intensive care unit.
- Administer levothyroxine 50–100 mcg IV daily.
- Administer gentle volume resuscitation for management of hypotension.
- Follow up all labs and cultures. Discontinue steroids if no adrenal insufficiency. Narrow antibiotic coverage as appropriate as culture data becomes available.
- Serial serum electrolytes with corrections as indicated.

Risk Factor

- Hypothyroidism

Prevention

Evaluate and treat hypothyroidism when it becomes symptomatic.

Special Considerations

- Controversy exists regarding the added benefit of administering T_3 in addition to levothyroxine.
- Most cases of myxedema coma occur during the winter in women >60 years old.

Further Reading

Wartofsky L. Myxedema coma. *Endocrinol Metab Clin North Am.* 2006; 35(4): 687–698.

Pheochromocytoma

Definition

A catecholamine-secreting chromaffin cell tumor usually found in the adrenal medulla.

Presentation

- Sustained or paroxysmal hypertension, tachycardia, and tachydysrhythmias that worsen and become more frequent with time
- History of headaches, chest pain, palpitations, and diaphoresis
- Myocardial ischemia
- Acute crisis may occur during induction of anesthesia or surgical manipulation of the tumor.

Pathophysiology

Excessive catecholamines secreted by a chromaffin cell tumor cause tachycardia and vasoconstriction. Cardiac manifestations

may be caused by increased myocardial oxygen demand or catecholamine-induced myocarditis.

DIFFERENTIAL DIAGNOSIS

- Light anesthesia
- Malignant hyperthermia (fever, mixed respiratory and metabolic acidosis)
- Malignant hypertension
- Alcohol withdrawal
- Illegal drug use: cocaine, amphetamines, PCP, LSD
- Pharmacologic agents: Monoamine oxidase inhibitors, decongestants, sympathomimetics
- Thyrotoxicosis (fever, acidosis, tachycardia)

Immediate Management

- If the patient is anesthetized, administer additional opioids or potent volatile anesthetics to decrease the blood pressure via vasodilation and myocardial depression.
- Return the blood pressure to a safe level:
 - Insert an intra-arterial catheter to monitor blood pressure.
 - Infuse sodium nitroprusside (starting dose is 1 mcg/kg/min), fenoldopam infusion (0.2–0.8 mg/kg/min), or nicardipine infusion (start at 5 mg/h).
 - Administer labetalol (20–40 mg IV every 10 minutes), or esmolol (500 mcg/kg IV, then 50 mcg/kg/min titrated to heart rate). **Use beta-blocking agents to control heart rate only after blood pressure is adequately controlled.** Betablockade combined with unopposed alpha-adrenergic activity may lead to severe hypertension and vasoconstriction.
 - Titrate medications to return the blood pressure to the patient's baseline.
- Administer intravenous fluids as necessary.
- Discontinue surgery as soon as practical.
- If myocardial ischemia refractory to beta-blockade is suspected, nitroglycerine should be used with caution.

Diagnostic Studies

- Plasma-free metanephrine and urinary fractionated metanephrine (highest sensitivity)
- Urinary vanillylmandelic acid (highest specificity)
- Computed tomography scan or MRI to evaluate to visualize adrenal mass
- Toxicology screening to rule out cocaine or other compounds

Subsequent Management

- Careful postoperative blood pressure monitoring (consider transfer to the intensive care unit)
- Consider Mg^{2+} (40–60 mg/kg bolus), then infusion 2 g/h. (Magnesium inhibits catecholamine release and has antiarrhythmic and vasodilator effects.)
- Consult endocrinology to confirm the diagnosis.
- Obtain a 24-hour urine collection to measure free catecholamines.

Risk Factors

- Associated with multiple endocrine neoplasia (MEN) type 2 (pheochromocytoma, medullary thyroid carcinoma, hyperparathyroidism), von Hippel-Lindau syndrome, or neurofibromatosis type 1.

Prevention

- Careful preoperative workup in patients with unexplained symptoms that are suggestive of pheochromocytoma.
- Careful manipulation of the adrenal glands in patients who have a known pheochromocytoma.

Special Considerations

- Hydralazine causes reflex tachycardia and should not be used as a sole antihypertensive.
- Labetalol is a vasodilator in addition to its cardiac effects and is preferred over metoprolol or other cardio-specific beta-blockers.
- Reducing preload with venodilation or attempted diuresis may be ineffective in pheochromocytoma patients. These patients generally have reduced intravascular volume due to renal excretion of both salt and water.

Further Reading

Connery LE, Coursin DB. Assessment and therapy of selected endocrine disorders. *Anesthesiol Clin North Am*. 2004; 22(1): 93–123.

Pacak K. Preoperative management of the pheochromocytoma patient. *J Clin Endocrinol Metab*. 2007; 92(11): 4069–4079.

Porphyria

Definition

A family of related enzyme disorders of heme pathway intermediates that cause pathologic accumulation of porphyrins or porphyrin

precursors. The disorders are classified as erythropoietic (bone marrow) or hepatic (liver) depending on where the accumulation occurs. The three most common types are porphyria cutanea tarda, erythropoietic porphyria, and acute intermittent porphyria (AIP). Acute intermittent porphyria is most likely to cause complications in the perioperative period.

Presentation

- Porphyria cutanea tarda: skin blistering related to sun exposure.
- Erythropoietic porphyria: skin lesions, occasional hemolysis, and rarely liver failure.
- Acute intermittent porphyria:
 - Abdominal pain (possibly related to autonomic neuropathy)
 - Sympathetic hyperactivity with hypertension and tachycardia
 - Central nervous system (CNS) symptoms, including confusion, hallucinations, seizures, and autonomic neuropathy
 - Progressive motor neuropathy that may lead to acute postoperative respiratory failure and reintubation. A patient who has received neuromuscular blocking drugs may fail to regain motor function after a long procedure.
 - Reddish-colored urine that darkens after exposure to light occurs during an acute attack.

Pathophysiology

The heme precursors are believed to be neurotoxic; delta-aminolevulinic acid and porphobilinogen are two characteristic intermediates in the heme biosynthetic pathway that are increased during acute attacks of porphyria.

DIFFERENTIAL DIAGNOSIS

- Acute abdomen
- Guillain-Barré syndrome

Immediate Management

- Withdraw any triggering agents, including barbiturates and ketamine.
- Begin aggressive hydration with dextrose-containing fluids.
 - Administer a bolus of D₅₀W. Begin D₁₀W at 1 mL/kg/h to deliver at least 3 L per day.
- Anticipate cardiovascular instability.
 - Consider beta-blockade (treats hypertension and tachycardia and may decrease ALA synthetase activity).
- Control pain with opiates.
- Treat nausea/emesis with phenothiazines.

Diagnostic Studies

- Urine porphobilinogen (diagnostic of an acute attack).
- Follow serum Na^+ , K^+ , and Mg^{2+} levels during acute attack.
- Measure porphyrin precursors and porphyrins in red blood cells, plasma, and/or urine will help to make the diagnosis in the OR or in the postanesthesia care unit.

Subsequent Management

- Admit patients with acute porphyria.
- Administer hemin (Panhemin, Lundbeck, Inc., Deerfield, IL) 3–4 mg/kg delivered as a single daily dose. Large-bore peripheral or central IV administration is preferred.
- Genetic testing is helpful for diagnosis confirmation and family analysis, but is not useful in the acute setting.
- A hematology consultation is strongly recommended for acute diagnosis and management.

Risk Factors

- Potential triggers (**not a complete list**): Alcohol, angiotensin converting enzyme inhibitors, anticonvulsants except gabapentin, barbiturates, calcium channel blockers (**especially nifedipine**), ergots, etomidate, progesterone, sulfonamide antibiotics
- Reduced caloric intake (especially carbohydrates)
- Dehydration

Prevention

- Avoid use of known triggering agents. A porphyria drug database is available at <http://www.porphyrifoundation.com/drug-database>.
- Patients with a family history of porphyria should undergo plasma volume expansion prior to elective procedures.
- Consider regional anesthesia if feasible and after evaluating the patient's neurological status. There is no evidence to suggest that local anesthetics can trigger an acute attack.

Special Considerations

- Propofol and benzodiazepines are probably safe.
- Inhaled anesthetics, including N_2O , are considered safe.
- Neuromuscular blocking agents (including succinylcholine) are widely considered to be safe but should be used with caution.
- Morphine, fentanyl, and sufentanil are safe.

- In general, a single exposure to even potent inducers may be tolerated, but repeat exposure or use during an acute attack is considered to be unsafe.
- Acute blood loss does not seem to provoke a porphyric attack.
- An attack may last for days, but is generally followed by complete recovery.
- A missed diagnosis of porphyria may lead to a nontherapeutic laparoscopy or laparotomy, although frank peritonitis is quite rare.
- Anhematin restores normal hepatic heme levels and suppresses aminolevulinic acid (ALA) synthetase activity.

Further Reading

James MF, Hift RJ. Porphyrias. *Br J Anaesth.* 2000; 85(1): 143–153.

Thyroid Storm

167

Definition

An acute, life-threatening, hypermetabolic state characterized by high levels of circulating catecholamines that is driven by excessive release of thyroid hormones.

Presentation

- Hyperthermia
- Tachycardia
- Hypertension
- Tremors
- Anxiety
- Delirium
- High fever (as high as 40–41° C)
- Diaphoresis

Pathophysiology

Thyroid storm is a state of severe sympathetic overactivity in the setting of clinical hyperthyroidism, generally precipitated by physiologic stress such as surgery or infection.

DIFFERENTIAL DIAGNOSIS

- Malignant hyperthermia (mixed metabolic and respiratory acidosis, creatine kinase is elevated in MH)
- Pheochromocytoma (no fever)

Immediate Management

- Establish large-bore peripheral or central venous access.
- Begin fluid resuscitation.
- Administer beta-blockers: labetalol 20–40 mg IV every 10 minutes, metoprolol 5 mg IV every 10 minutes, or esmolol 80 mg over 5 minutes, then 150 mcg/kg/min infusion.
- Administer glucocorticoids (possibility of adrenal insufficiency; glucocorticoids block conversion of T_3 to T_4): dexamethasone 4 mg IV every 6 hours or hydrocortisone 100 mg IV every 6 hours.
- Supplement Mg^{2+} as needed.
- Administer acetaminophen or NSAIDs to decrease fever.

Diagnostic Studies

- Thyroid function studies including T_3 , free T_4 , TSH
- Serum electrolytes
- Electrocardiogram

Subsequent Management

- If the patient is already intubated, consider continuing mechanical ventilation into the postoperative period.
- Administer a loading dose of antithyroid medication: propylthiouracil (PTU) [200 mg every 4 hours] or methimazole [20 mg every 6 hours]. (Both are oral preparations.)
- Begin administration of elemental iodine after starting the antithyroid medication.
- Consult the endocrinology service to guide subsequent management.

Risk Factors

- Recent iodine therapy (radioiodine or iodine-containing contrast agents)
- Cessation of antithyroid medications
- Infection or serious illness (e.g., myocardial infarction) in a patient with pre-existing hyperthyroidism
- Trauma
- Pre-eclampsia
- Excessive thyroid hormone replacement
- Excessive manipulation of a hypertrophic thyroid gland during surgery

Prevention

Establish an adequate depth of anesthesia in a patient with hyperthyroidism who is undergoing surgery in order to avoid an excessive sympathetic response.

Special Considerations

- Emergency therapy consists of circulatory support and blockade of the end-organ effects of the excess thyroid hormone.
- Intraoperative hypotension should be treated with a direct-acting vasoconstrictor (e.g., phenylephrine).

Further Reading

Connery LE, Coursin DB. Assessment and therapy of selected endocrine disorders. *Anesthesiol Clin North Am.* 2004; 22(1): 93–123.

Transurethral Resection of the Prostate Syndrome

169

Definition

Systemic absorption of fluids used for bladder distention during a transurethral resection of a prostate mass (TURP). Occurs in approximately 2% of patients undergoing this procedure.

Presentation

- Hyponatremia
- Central nervous system manifestations:
 - Mental status changes
 - Diplopia
 - Nausea and vomiting
- Cardiovascular symptoms:
 - Hypertension
 - Bradycardia
 - Myocardial ischemia and dysrhythmia

Pathophysiology

Nonionic irrigating solution (mannitol, glycine, or sorbitol) enters the circulation through open venous sinuses. Excessive absorption of irrigating solution leads to hyponatremia, circulatory overload, or neurotoxicity (if glycine-containing solution is used).

DIFFERENTIAL DIAGNOSIS

- Congestive heart failure
- Hyponatremia

Immediate Management

- Discontinue administration of irrigation fluid.
- Send blood for serum electrolyte levels (Na^+).
- Administer furosemide (20 mg IV); adjust dose for preoperative creatinine.
- The TURP syndrome is usually time-limited (generally resolves within 6 hours after surgery).
- Terminate the procedure as soon as practical.

Diagnostic Studies

- Complete blood count
- Basic metabolic panel

Subsequent Management

- Continue monitoring into the postoperative period until symptoms abate. The patient may be monitored in the postanesthesia care unit or the intensive care unit.
- Consider administration of hypertonic (3%) saline in patients with severe hyponatremia (<120 mEq/L, or those with neurologic symptoms).

Risk Factors

- Prolonged operative time (approximately 1 liter of fluid is absorbed per 40 minutes operating time)
- High irrigating pressure (determined by the height of the bag above the patient)
- Large prostate, extensive resection
- Glycine-containing solution (may cause transient blindness)

Prevention

- Consider use of regional anesthesia (permits early detection of CNS changes).
- The patient should be kept horizontal, because using the Trendelenburg position reduces the intravesical pressure required to initiate absorption, thus increasing the risk of irrigation fluid absorption.
- Avoid glycine-containing solutions if able.
- The height of the irrigating fluid bag should be approximately 60 cm above the patient.

Special Considerations

- There are no definite criteria to diagnose TURP syndrome. The clinician must have a high index of suspicion.
- Use of glycine-containing solution may cause transient blindness.

Further Reading

Malhotra V. Transurethral resection of the prostate. *Anesthesiol Clin North Am.* 2000; 18(4): 883–897.

Chapter 7

Neurosurgical and Neurologic Emergencies

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Autonomic Hyperreflexia or Dysreflexia	174
Intracranial Hypertension	175
Spinal Cord Injury	178
Subarachnoid Hemorrhage	181
Traumatic Brain Injuries: Closed and Open Traumatic Injuries	184
Venous Air Embolism	187
Special Considerations	189

Autonomic Hyperreflexia or Dysreflexia

Definition

An abrupt and profound autonomic discharge from sympathetic neurons caused by stimulation, such as distention of hollow viscous, distal to the level of a spinal cord injury resulting in severe hypertension and an impending medical emergency.

Presentation

- In an awake patient, autonomic hyperreflexia causes an abrupt onset of headache and visual changes, such as blurred vision or scotoma, consistent with a rapid and significant elevation in blood pressure.
- Elevated blood pressure may cause a reflex baroreceptor-mediated bradycardia.
- In either an asleep or awake patient, skin erythema, diaphoresis, and/or piloerection above the dermatome of spinal injury may also occur.

Pathophysiology

As little as 2 weeks after injury to the spinal cord, descending control of preganglionic sympathetic neurons below the level of injury is lost. Uninhibited spinal circuits can then produce massive sympathetic discharge after stimulation from bladder distention, bowel distention, or skin incision. The discharge causes release of catecholamines, dramatically elevated blood pressure, and reflex bradycardia in 50% to 70% of individuals with cord injury at or above T6. Spinal anesthesia can decrease the risk of sympathetic discharge.

DIFFERENTIAL DIAGNOSIS

- Light anesthesia
- Untreated chronic hypertension
- Pheochromocytoma

Immediate Management

- Place patient in the head-up position if possible.
- Immediately discontinue surgical stimulation and release the pressure in a distended hollow viscus (e.g., empty the bladder)
- If the patient is awake, direct him or her to “bite and swallow” a nifedipine capsule.
- In the anesthetized patient, deepen anesthesia and consider beginning intravenous nitroglycerine (20–40 mcg bolus at 3- to 5-minute intervals or 0.5 mcg/kg/min) or nitroprusside (3–4 mcg/kg/min titrated to effect up to 10 mcg/kg/min) to rapidly treat dangerous systolic hypertension.

Subsequent Management

If subsequent procedures are required, prophylaxis may be indicated. Oral therapy with prazosin (1–3 mg by mouth two or three times per day) or phenoxybenzamine (10–40 mg by mouth two or three times per day) may reduce the risk for later events.

Risk Factors

- Spinal cord injury above T6 with distension of hollow viscous
- Male gender (four times more likely than females)

Prevention

If feasible, spinal anesthesia with local anesthetic can be used to prevent sympathetic discharge with stimulation.

Further Reading

Barash PG, Cullen BF, Stoelting RK, Cahalan MK, Stock MC, Ortega J, eds. *Clinical anesthesia*. 7th ed. Philadelphia: Lippincott Williams & Wilkins; 2013:1024.

Bycroft J, Shergill IS, Chung EA, Arya N, Shah PJ. Autonomic dysreflexia: a medical emergency. *Postgrad Med J*. 2005; 81(954): 232–235.

Intracranial Hypertension

Definition

Intracranial hypertension (ICH) in adults is defined as an elevation in ICP >15 mm Hg. Values above 20–25 mm Hg generally require intervention to reverse symptoms and restore cerebral perfusion. Sustained values >25 mm Hg cause significant injury, and values >40 mm Hg are life threatening. In general, normal values are considered to be 10–15 mm Hg for adults, 4–7 mm Hg for young children, and 1.5–6 mm Hg for full-term infants.

Presentation

- In the awake patient, symptoms include headache, visual changes, nausea, and vomiting.
- As ICH is sustained or increases, alterations in mental status, with confusion and decreased responsiveness occur.
- Cushing's triad of elevated blood pressure, reflex bradycardia, and irregular respiration can be observed with ICH and are late and ominous signs.
- Papilledema can be observed with ICH, but its absence does not rule out the presence of ICH.

- Pupillary dilation unreactive to light and decorticate posturing are associated with brain herniation; they are late and ominous signs.

Pathophysiology

Intracranial contents consist of brain tissue, cerebrospinal fluid (CSF), and blood contained within the non-compliant skull. Because the noncompliant skull does not allow for significant increases in volume, any increase in volume can increase pressure, causing ICH. Physiologic causes include increased abdominal or thoracic pressures (e.g., a Valsalva maneuver), head down position, seizure, hypoventilation, and some drugs (e.g., high-dose potent volatile anesthetics). Pathologic causes include intracranial hemorrhage, cerebral edema, tumor, and disturbances in CSF production, flow, or absorption. The rate at which the volume of blood, tissue, or CSF changes is critical. When the volume of one component changes rapidly, there is less time for compensation. Therefore, rapid changes are tolerated poorly. Very high ICP may ultimately cause herniation, in which parenchyma is displaced across the falx cerebri, the tentorium cerebelli, or the foramen magnum. Herniation generally occurs late and is associated with poor prognosis.

DIFFERENTIAL DIAGNOSIS

- Headache of other etiology
- Drug intoxication
- Idiopathic intracranial hypertension (pseudotumor cerebri)

Immediate Management

- Determine whether circulatory support and airway management are needed. (Patients with ICH may be unable to control their airway.)
- Place the patient in the “head up” position of 30°–40° and facing straight ahead to prevent jugular venous occlusion.
- Administer mannitol 20% 0.5–1.0 g/kg IV or hypertonic saline (3%–7.2% with total of 7–9 g given over 30 minutes to 1 hour) to decrease brain water content and ICP.
- Control systemic blood pressure with phenylephrine (50–100 mcg boluses at 1- to 2-minute intervals or 100 mcg/min infusion), ephedrine (5–10 mg boluses), or labetalol (10-mg boluses at 1- to 2-minute intervals) to with a goal cerebral perfusion pressure of 60–70 mm Hg.
- Provide sedation and analgesia as appropriate.
Note: Reversible or short-acting drugs are preferred. Monitor the patient for signs of respiratory depression.

Immediate Management (continued)

- Control fever if present.
- If ICH continues, intubate the trachea and initiate mechanical ventilation to induce mild cerebral vasoconstriction and reduce ICP as a bridge to definitive surgical treatment.
- Consider seizure prophylaxis (e.g., Levetiracetam. Loading dose is 10 mg/kg IV over 15 minutes).
- Maintain close communication with the neurosurgical team regarding imaging results and need for invasive intracranial pressure (ICP) monitoring, CSF drainage, or emergency craniotomy.

Diagnostic Studies

- Computed tomography (CT) scan to diagnose surgical causes (e.g., hematoma, tumor), assess ventricular size, compression of basal cisterns, mass effect, edema.
- Invasive ICP monitoring
- Lumbar puncture to measure opening pressures (if CT scan shows absence of mass effect)
- Note: Diagnostic studies should delay an urgent surgical intervention; coagulation panel should be obtained if immediate reversal of anticoagulation medications is required.

Subsequent Management

- Identify and correct the underlying cause of ICH. Obtain a neurosurgical consultation if imaging is suggestive of a surgically correctible etiology.
- Control blood glucose to avoid hypoglycemia and hyperglycemia to improve outcomes.
- If required, administer continued ventriculostomy drainage and ICP monitoring.

Risk Factors

- Intracranial hemorrhage
- Severe serum hyponatremia or hypo-osmolality causing edema
- Malignancy: primary or metastatic
- Traumatic injury
- Hydrocephalus
- Infection (e.g., brain abscess)
- Congenital anomalies

Special Considerations

- Rapid recognition and initiation of treatment is critical for improving neurologic outcome in patients with ICH.

Further Reading

Rangel-Castillo L, Gopinath S, Robertson CS. Management of intracranial hypertension. *Neurol Clin.* 2008; 26(2): 521–541.

Ruskin, KJ, Rosenbaum SH, Rampil IJ, eds. *Fundamentals of neuroanesthesia.* 1st ed. Stony Brook, NY: Oxford University Press; 2014:16–25.

Spinal Cord Injury

Definition

Damage to spinal tissue caused by trauma, ischemia, hematoma, tumor, or other insult that causes temporary or lasting loss of sensory, motor, and/or autonomic function. The clinical signs and symptoms depend on the type and level of cord injury, ranging from paresthesias to complete sensory, motor, and autonomic paralysis.

Presentation

- Quadraplegia with incomplete or complete loss of motor function is caused by a lesion at the level of the cervical spinal cord. Patients with C3 injury or above almost always require airway management and mechanical ventilation; C3–5 injuries vary in the degree of phrenic nerve paralysis.
- Paraplegia with incomplete or complete loss of motor function of legs, torso, or pelvic organs is caused by injury to the thoracic cord. Motor function of the arms is preserved. Higher thoracic level injuries (T8 and above) may cause weakened intercostal muscles and affect coughing ability.
- *Neurogenic shock* from loss of autonomic input from the thoracic spine causes hypotension, bradycardia, and hypothermia.
- *Spinal shock* with loss of reflexes and motor function below the level of injury.
- *Anterior spinal artery syndrome* presents with loss of pain and temperature with preservation of vibration, touch, and proprioception due to interruption of blood flow through the anterior spinal artery.
- *Brown-Sequard syndrome* is defined as ipsilateral loss of motor function, vibration, touch, and proprioception with contralateral loss of temperature and pain sensation secondary to hemi-cord injury.

- *Cauda equina syndrome* presents with loss of bowel and bladder control and leg weakness caused by compression of the bundle of lumbosacral nerve roots.
- *Central cord syndrome* presents with loss of motor function in the upper extremities greater than loss in lower extremities, and a variable loss of sensory function below the level of injury secondary to injury of the central portion of the spinal cord.

Pathophysiology

Primary injury to the spinal cord can be caused by compression, transection, or traction from hematoma, disk subluxation, hyperextension, or fractures of bony elements in the spinal column. Secondary injury results from interruption of vascular supply and tissue ischemia from edema and inflammation after the primary injury. Secondary injury may cause the injury to involve higher cord levels, resulting in an evolving group of signs and symptoms. Injuries are divided into five classes based on impairments:

- Complete transection with no sensory or motor function preserved in sacral segments S4-S5
- Incomplete preservation of sensory but not motor function below the injured level, including sacral segments S4-S5
- Incomplete preservation of motor function below the injured level with more than half of the muscles below the injury with strength $<3/5$
- Incomplete preservation of motor function below the injured level with more than half of muscles below injury with strength $\geq 3/5$
- Sensory and motor function are not impaired.

DIFFERENTIAL DIAGNOSIS

- Soft tissue injury of neck causing muscle spasm
- Conversion motor paralysis disorder

Immediate Management

- Assess the patient's airway, breathing and circulation. Note: If intubation of the trachea is indicated in a patient with a cervical spine injury, a technique that maintains spinal alignment (e.g., in-line cervical spine stabilization or fiberoptic intubation) must be used.
- Avoid hypoxemia; provide supplemental oxygen as required.
- Rapidly examine the patient to identify additional life-threatening injuries.
- Treat hypotension caused by neurogenic shock with vasopressors, phenylephrine (50- to 100-mcg boluses at 1- to

Immediate Management (*continued*)

2-minute intervals or 100 mcg/min infusion), or ephedrine (5- to 10-mg boluses). This condition is relatively resistant to fluid resuscitation.

- Administer steroids, such as methylprednisolone (30 mg/kg IV followed 5.4 mg/kg/h for 24 hours). This has been shown to reduce cord edema and improve outcome. Note: The specific protocol to be used should be discussed with the neurosurgery team if possible.
- Therapeutic hypothermia is not recommended at this time.
- Maintain close communication with the neurosurgical team regarding imaging results and the need for emergency surgical decompression.

Diagnostic Studies

- Rapid total body CT to diagnose cord injury and other associated injuries; CT images are reformatted to assist in immediate diagnosis of injuries to vertebral column and spinal cord.
- Depending on the stability of the patient, magnetic resonance imaging is useful to assess soft tissue and cord injury.
- Note: Diagnostic studies should not delay emergency surgical intervention.

Subsequent Management

- If subsequent procedures requiring anesthesia are needed, avoid succinylcholine in the period 24–72 hours after injury to avoid the development of life-threatening hyperkalemia.
- Begin venous thromboembolism prophylaxis.
- Pressure ulcer prophylaxis to prevent decubitus ulcers.
- Consider management to prevent neuropathic pain when the patient is stable.
- Control hypothermia.

Risk Factors

- Male gender
- Younger age
- Risk-taking behavior
- Elderly patients with increased risk for falling

Prevention

Avoid risk-taking behaviors. Modify risks in elderly with significant potential to fall.

Further Reading

- Bonner S, Smith C. Initial management of acute spinal cord injury. *Cont Educ Anaesthes Crit Care Pain*. 2013.
- Bracken MB. Steroids for acute spinal cord injury. *Cochrane Database Syst Rev*. 2012; Jan 18; CD001046.
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- Miko I, Gould R, Wolf S, Afifi S. Acute spinal cord injury. *Int Anesthesiol Clin*. 2009; 47(1): 37–54.

Subarachnoid Hemorrhage

Definition

Subarachnoid hemorrhage (SAH) is a cerebrovascular accident in which bleeding occurs into the subarachnoid space. Extravasation of blood into the CSF can be caused by trauma or from nontraumatic bleeding of vascular defects, such as arteriovenous malformation (AVM) or from an arterial aneurysm.

Presentation

- Sudden onset, severe, “thunderclap” headache is a classic sign of SAH. “Worst headache of my life.”
- Nausea and vomiting are common.
- Confusion or agitation, decreased level of responsiveness, or transient loss of consciousness may occur.
- Nuchal rigidity may be present.
- Photophobia and seizure may occur.
- Hypertension and elevated temperature are common signs.
- Focal deficits from ischemia, such as cranial nerve palsy, may occur.

Pathophysiology

Seventy to eighty-five percent of SAH events are secondary to aneurysmal bleeding, with the remainder of events caused by AVM, tumor, infection, or trauma. Arterial bleeding can rapidly increase ICP, potentially causing devastating neurologic impairment.

DIFFERENTIAL DIAGNOSIS

- Headache of other etiology
- Tumor
- Infection or abscess

Immediate Management

- Assess the patient's airway, breathing, and circulation. Intubate the trachea and initiate mechanical ventilation if indicated.
- Obtain large-bore peripheral IV access and insert an intra-arterial catheter for blood gas analysis and continuous blood pressure monitoring
- Stabilize the blood pressure to achieve a CPP 60–70 mm Hg using vasopressors (e.g., phenylephrine 0.5- to 1-mcg/kg/minute infusion or 100-mcg boluses) or intravenous short-acting beta blockers (e.g., labetalol) and/or calcium channel blockers (e.g., nicardipine; starting dose 5 mg/hour).
- Consider moderate hypocapnia (PaCO_2 25–30 mm Hg) to reduce ICP until definitive treatment is achieved. Note: Hypercapnia decreases cerebral blood flow and worsens cerebral ischemia. This should only be used as a bridge to definitive treatment (e.g., surgical decompression).
- Maintain euvolemia.
- Mild hypothermia is not indicated and does not improve neurologic outcome.
- Request a consultation from the neurosurgical team to plan definitive therapy, which may include intravascular therapy (i.e., coiling or embolization) or surgical management (i.e., aneurysm clipping or AVM resection).

Diagnostic Studies

- Computed tomography scan without contrast, CT angiography: The Fisher grading scale, which describes the amount of blood observed on CT, helps to predict the occurrence of cerebral vasospasm.
- Plasma electrolytes, complete blood count, coagulation profile, arterial blood gas analysis
- Blood type and cross-match
- Direct intravascular angiography
- Electrocardiogram (often reveals repolarization abnormalities)
- Note: Diagnostic studies should not delay urgent surgical intervention.

Subsequent Management

- Close monitoring of neurologic status is imperative because re-bleeding may occur and carries a poor prognosis.
- If vasospasm occurs, consider treatment with prophylaxis with nimodipine (60 mg by mouth or via nasogastric tube every 4

hours for 21 days), hypertensive hypervolemic hemodilution (“triple-H” therapy). Intravascular angioplasty, or stenting also should be considered.

- A ventricular drain may be required for patients who develop obstructive hydrocephalus after SAH.
- Additional management is guided by systemic complications, which catecholamine-induced cardiac abnormalities (e.g., dysrhythmia, myocardial infarction, heart failure), pulmonary hypertension and edema, and electrolyte disturbances (e.g., hyponatremia caused by syndrome of inappropriate anti-diuretic hormone [SIADH]).

Risk Factors

- Uncontrolled hypertension
- Smoking
- Alcohol use
- Family history of aneurysm

Special Considerations

- Subarachnoid hemorrhage accounts for approximately 10%–15% of all strokes. Intraparenchymal hemorrhage is not considered SAH. The majority of aneurysms are not familial, but are instead associated with risk factors of uncontrolled hypertension, alcohol use, and smoking. Most ruptures are spontaneous. Sites of rupture are well defined, most commonly at the anterior communicating arteries, followed by posterior communicating/internal carotid arteries, then followed by the middle cerebral artery. The Hunt and Hess scale and the grading system of the World Federation of Neurological Surgeons are both acceptable predictors of morbidity and mortality after SAH. Vasospasm is a common and devastating complication after SAH.

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Traumatic Brain Injuries: Closed and Open Traumatic Injuries

Definition

An acute traumatic injury to the head and skull carrying a sudden concussive or shearing force transmitted to brain tissue. In addition to traumatic force, open traumatic injuries also involve fractures or penetration of the skull.

Presentation

- Head injuries are *widely variable* in severity, mechanism, and presentation, depending on the nature of the injury sustained.
- In the awake patient, one may elicit a history of sudden impact or acceleration/deceleration injury, followed by brief loss of consciousness. Other symptoms may include amnesia, confusion, headache, photophobia, or hemiparesis.
- Individuals may exhibit lethargy or decreased responsiveness, with decreased pupillary light reflex.
- Post-traumatic seizures may be present.
- Patients with severe injuries may present with cardiovascular instability, including bradycardia, other dysrhythmia, and hypotension.
- Epidural hematomas are associated with a severe head impact. The classical presentation consists of a brief loss of consciousness, followed by a lucid interval, followed by rapid progression to decreased level of consciousness and coma.
- Subdural hematomas, traumatic subarachnoid hemorrhage, and intraparenchymal hemorrhage each can result from traumatic injuries, but typically do not have a lucid interval.

Pathophysiology

Traumatic brain injury is widely variable in type, severity, and associated injuries. *Primary injury* includes direct injury of brain parenchyma from contusion, shearing, or penetrating injury from bone or foreign body. *Secondary injury* is defined as tissue damage that is caused by ischemia, edema, and inflammation that leads to transient or more permanent brain dysfunction. Secondary injury is exacerbated by vascular injury, hypoventilation, and associated cardiovascular events with or without intracranial hypertension (ICH).

DIFFERENTIAL DIAGNOSIS

- Rupture of pre-existing arteriovenous malformation or aneurysm
- Chronic subdural hematoma

Immediate Management

- Assess the patient's airway, breathing, and circulation. Intubate the trachea and initiate mechanical ventilation if indicated. Note: If intubation of the trachea is indicated in a patient with a cervical spine injury, a technique that maintains spinal alignment (e.g., in-line cervical spine stabilization or fiberoptic intubation) must be used.
- Provide supplemental oxygen if needed to avoid hypoxemia.
- Rapidly examine the patient to identify additional life-threatening injuries.
- Resuscitate the patient with normal saline or, if indicated, blood products. (Note: If massive transfusion is indicated for other injuries, consider infusing packed red blood cells, platelets, and fresh-frozen plasma in a 1:1:1 ratio.)
- The cerebral perfusion pressure should be at least 60 mm Hg. Use vasopressors as necessary to achieve this goal (e.g., phenylephrine 100 mcg IV bolus or 0.5–1 mcg/kg/min). If ICH is suspected, increase the systemic blood pressure as needed to maintain CPP.
- Administer mannitol 0.5–1.0 g/kg if ICH is suspected in the absence of disruption of blood-brain barrier.
- If required, adjust mechanical ventilation to maintain PaCO_2 30–35 mm Hg.
- Request a consultation from the neurosurgic team to plan definitive therapy, including the need for ICP monitoring, CSF drainage, or emergency craniotomy.

Diagnostic Studies

- Plasma electrolytes, complete blood count, coagulation profile
- Arterial blood gas analysis as necessary to guide ventilator management
- Computed tomography of the brain with and without contrast; cervical spine CT (to rule out concomitant cervical spine injury)
- Worsening symptoms or level of critical illness will guide the clinician's need for further diagnostic studies.
- Note: Diagnostic studies should not delay urgent surgical intervention.

Subsequent Management

- Continue supportive care.
- Periodically re-evaluate the patient for injury progression; changes in neurologic status guide diagnostic studies and surgical referral.

- Consider insertion of a ventriculostomy catheter to facilitate CSF drainage and control of ICH.
- If appropriate, early referral to rehabilitation offers the patient the best chance for improvement of neurologic deficits resulting from the injury.

Risk Factors

- Younger age and male gender
- Pre-existing coagulopathy increases risk of delayed neuronal deficits.
- Age >60 years increases risk of SDH.

Prevention

Avoidance of risk-taking behavior

Special Considerations

- Suspect ICH if the patient has an altered mental status or symptoms such as headache, nausea, vomiting, or visual changes.
- Suspect an epidural hematoma if the patient presents with a depressed skull fracture in the temporal-parietal region with trauma to the middle meningeal artery.
- Subdural hematomas are classified as acute or chronic; acute hemorrhage is more likely to progress quickly to ICH and neurological deterioration. A chronic subdural hematoma progresses more slowly and is less likely to result in acute changes in ICP.
- Glucose-containing solutions should be avoided to reduce hyperglycemia, which is associated with poorer outcomes.
- Guidelines for surgical decompression include midline shift >5 mm, fixed pupils, ICP >20 mm Hg, hematoma thickness >1 cm, or severe mental status impairment.
- High-dose corticosteroids and mild hypothermia have not been shown to improve outcomes and are not indicated.
- Progesterone holds promise for improving neurologic outcome following traumatic brain injury, but is still considered experimental at this time.

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Venous Air Embolism

Definition

Venous air embolism (VAE) is an iatrogenic, potentially life-threatening emergency caused by the entrainment of atmospheric air into the systemic venous system.

Presentation

- In an awake patient, such as during central line placement, early signs include coughing, chest pain, and possibly new neurologic deficits.
- If air is entrained slowly, subtle changes in vital signs may be observed, including hypotension, tachycardia, or bradycardia. In a mechanically ventilated patient, peak airway pressure may be increased. Hypoxia may be seen on pulse oximetry or arterial blood gas analysis; hypercarbia may be noted on arterial blood gas analysis.
- If a large volume of air is entrained, an abrupt decrease in end-tidal CO_2 will be observed, coupled with an increase in PaCO_2 .
- Dysrhythmias, myocardial ischemia, right heart strain, and cardiovascular collapse may occur if significant air enters circulation.
- The surgeon may note the presence of gas bubbles in the arterial blood supply.

Pathophysiology

During a surgical procedure or after trauma, veins are exposed allowing communication between the atmosphere and circulation. Air typically enters the venous circulation through open venous structures above the level of the heart. Air trapped in the right heart and pulmonary vasculature may then cause an “air-lock” and cardiovascular collapse as venous return decreases, or air may travel into the systemic circulation through right-to-left shunting in the heart, causing cerebral or myocardial ischemia.

DIFFERENTIAL DIAGNOSIS

- Tension pneumothorax
- Thrombotic pulmonary embolism
- Ventricular dysrhythmia or myocardial infarction of other etiology

Immediate Management

- Alert the surgeon to the suspected diagnosis of VAE. Ask him or her to flood the field with saline to inhibit further air entrainment.
- Increase FiO_2 to 100%.
- If possible, place the patient in the head down (Trendelenburg) position.
- Support blood pressure with vasopressors (e.g., phenylephrine) or inotropes as needed.
- Administer isotonic crystalloid fluids to support blood pressure and increase right atrial pressure.
- If possible, moving the patient to the left-lateral decubitus position may relieve the air-lock in the right heart.
- If available, aspiration of air through a single- or multi-orifice catheter may decrease the amount of air in circulation.
- If cardiovascular collapse occurs, begin Adult Cardiac Life Support (ACLS).

Diagnostic Studies

- Transesophageal echocardiography is sensitive, and can detect both very small amount of air in circulation and right-to-left communication.
- Precordial Doppler ultrasound also is sensitive and can detect small amounts of air.

Subsequent Management

- If hemodynamically significant VAE occurs, a discussion with the surgeon regarding the continuation of the case is prudent.
- If right-to-left shunting of air embolus has occurred, hyperbaric oxygen therapy will decrease the air bubbles and improve oxygenation to ischemic tissues.
- Encourage liberal use of bone wax, soaking the field, and avoidance of large veins should be reiterated.

Risk Factors

- Venous air embolism classically occurs in sitting craniotomy
- Venous air embolism is a risk in many other surgical procedures, including but not limited to caesarean section, arthroscopic surgery of the shoulder, production of CO_2 tension pneumoperitoneum for laparoscopic surgery, central venous catheterization, thoracocentesis, chest trauma, high-pressure mechanical ventilation, and invasive vascular procedures.

Prevention

- Avoid sitting, head-up positions.
- Recommendation to surgeon of careful dissection of venous structures and judicious use of bone wax is critical.
- Preoperative fluid loading may increase CVP and avoid large pressure gradients.
- Use of positive end-expiratory pressure (PEEP) may increase CVP and decrease the incidence of VAE, but at the risk of increased risk of right-to-left shunting of bubbles.

Special Considerations

- The volume of entrained air during the event determines, in part, the severity of cardiovascular compromise the patient will suffer. Although the size of a VAE is difficult to estimate, volumes as small as 20 mL of air have caused significant hemodynamic changes in the adult. If a right-to-left intracardiac communication exists (e.g., through a patent foramen ovale), as little as 0.5 to 2 mL of air can cause stroke or myocardial infarction if it becomes lodged in cerebral or coronary vessels. A large volume of air may become trapped in the right heart, decreasing blood flow to pulmonary circulation and increasing strain on the right ventricle. There is an increase in pulmonary artery pressures and a decrease in venous return to the left heart, causing decreased cardiac output and impending cardiovascular collapse.

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Chapter 8

Obstetric Emergencies

Robert R. Gaiser

Accidental Dural Puncture	192
Diagnostic Studies	193
Breech Presentation	195
Embolism: Thrombus, Amniotic Fluid, Air	198
Failed Intubation	202
Fetal Bradycardia	205
Hypotension	210
Local Anesthetic Toxicity	213
Maternal Cardiac Arrest	216
Maternal Hemorrhage	219
Neonatal Resuscitation	223
Pre-eclampsia	226
Sepsis	230
Shoulder Dystocia	232
Total/High Spinal Anesthesia	234
Umbilical Cord Prolapse	237
Uterine Rupture	239

Accidental Dural Puncture

Definition

Accidental dural puncture occurs when the both the dura and subarachnoid maters are punctured unintentionally during epidural catheter placement.

Presentation

Any one of the following signs indicates accidental dural puncture:

- Free flowing cerebrospinal fluid (CSF) from the epidural needle
- Test dose of 3 mL 1.5% lidocaine with epinephrine 1:200,000 through the epidural catheter produces evidence of a sensory and motor block
- Cerebrospinal fluid is aspirated from the catheter
 - Cerebrospinal fluid may be distinguished from normal saline or local anesthetic by the presence of protein (15–45 mg/dL) and glucose (50–80 mg/dL).
- Presence of a frontal-occipital headache 24–48 hours after epidural anesthesia

Pathophysiology

The distance from the ligamentum flavum to the dura mater in the lumbar spine averages 4–6 mm. Dural puncture generally occurs when the person performing the procedure fails to recognize that the needle is in the epidural space or advances the local anesthetic infiltration needle too far. The hole in the dura and arachnoid maters makes it possible for local anesthetic to enter the subarachnoid space, causing high sensory and motor levels. Headache tends to occur 24–48 hours after accidental dural puncture and is caused by CSF leaking into the epidural space. The loss of CSF from the intrathecal space results in a decrease in intracranial pressure, causing traction on pain-sensitive structures, including the dura and meninges. This mechanism is proposed for the etiology of the headache. Other symptoms accompanying the headache include visual disturbances (diplopia) and hearing alteration, as cranial nerves III, IV, VI, and VIII are frequently involved.

DIFFERENTIAL DIAGNOSIS

Injecting local anesthetic into the subdural space (between the dura and arachnoid mater) may result in a high blockade that may also cause profound hypotension but differs by causing a patchy, weak block.

Immediate Management

- If the provider notes free-flowing CSF from the epidural needle, there are two courses of management:
 - Immediately remove the needle and redo the procedure at another location and slowly titrate the local anesthetic
 - This approach has the risk of a repeat accidental dural puncture.
 - Use caution when injecting local anesthetic through the resited epidural catheter
- Thread an epidural catheter intrathecally as rapidly as possible to limit loss of CSF. The catheter should be threaded only 3–4 cm into the intrathecal space. The catheter is then managed as continuous spinal analgesia.
 - Insertion of an intrathecal catheter does not prevent the development of a headache.
 - Limit the number of times the catheter hub is disconnected from the continuous infusion to prevent infection when using continuous spinal anesthesia.

193

Diagnostic Studies

Cerebrospinal fluid contains both protein (15–45 mg/dL) and glucose (50–80 mg/dL). Both of these may be measured in the laboratory. A simpler approach would be to use a dipstick for the presence of protein or glucose or to use a glucometer to measure the glucose concentration.

Subsequent Management

- If the epidural catheter was threaded intrathecally, it should be managed as a continuous spinal catheter.
 - The medication administered through an intrathecal catheter is the same as one would use for epidural analgesia, starting at a lower rate. The usual starting rate is 1–2 cc/h and adjusted based upon patient response.
- If the epidural space was relocated with the catheter threaded epidurally, the amount of local anesthetic used for the initial bolus should be decreased because it may leak through the hole into the dura and arachnoid maters.
- In both situations the patient should be evaluated daily for 48 hours and also should receive a follow-up phone call once discharged.

- If the patient develops a headache (risk factors include young age, female gender, and vaginal delivery), the patient should be offered an epidural blood patch. There is no value to conservative measures. Caffeine has not been shown to be beneficial.
- An epidural blood patch involves the injection of 20 mL of autologous blood into the epidural space. Blood compresses the thecal sac, forcing CSF cephalad; a clot forms at the dural hole preventing further leakage.

Risk Factors

- For accidental dural puncture
 - Previous accidental dural puncture—patients who had one accidental dural puncture are at increased risk for a second puncture, suggesting a possible anatomic cause.
- For postdural puncture headache
 - Young age (10–40 years)
 - Female gender
 - Vaginal delivery

194

Prevention

There are no effective means to prevent the development of a postdural puncture headache. A prophylactic epidural blood patch through the catheter is not effective. Inserting an intrathecal catheter decreases the risk of the patient experiencing a second puncture during relocating the epidural space, but does not decrease the incidence of headache. The use of cosyntropin has been investigated but has not gained widespread acceptance.

Special Considerations

- Cerebrospinal fluid may be aspirated either through the epidural needle or epidural catheter. Patients occasionally develop symptoms consistent with accidental dural puncture even though CSF is not aspirated. This may be due to a nick in the dura that was caused by either the epidural needle or the needle used for local anesthetic infiltration. The major concern after accidental dural puncture is postdural puncture headache, which tends to be bilateral, positional, and located in the frontal-occipital area. Neck stiffness and nausea may also occur. If the patient has an accidental dural puncture and becomes febrile in the postoperative period, meningitis must be strongly considered and diagnosed with lumbar puncture.

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Breech Presentation

Definition

Breech presentation is the fetal position in which the presenting part is not the head. A breech presentation occurs whenever the fetal buttocks descend into the maternal pelvis before the fetal head. Breech presentations are typically classified according to the position of the fetus within the uterus.

1. Frank breech: the fetal hips are both flexed and both knees are extended. This is the most common type of breech presentation and accounts for 70% of all breech presentations.
2. Complete breech: both the fetal hips and fetal knees are flexed. Complete breech accounts for 25% of all breech presentations.
3. Footling breech: one or both hips are extended. With the hip or hips being extended, one or both feet are lowermost in the birth canal. Footling breech is a more common presentation for premature fetuses rather than a term fetus and accounts for 5% of all breech presentations.

Pathophysiology

The etiology of breech presentation is unknown but is more likely when the fetus is premature or in a multigestation pregnancy. Women with a previous cesarean delivery have an increased incidence of breech presentation. A fetus found to be in the breech position prior to 24 weeks' presentation will most likely turn itself into a cephalic position.

The major concern with delivery of the breech fetus is fetal head entrapment. In a cephalic presentation, the obstetrician will be able to detect that the head is too large for the pelvis because labor will progress poorly or the fetus will not descend into the birth canal. A fetus in the breech presentation can deliver through a partially dilated cervix but the head will be

trapped. This causes compression of the umbilical cord against the cervix, which leads to decreased fetal blood flow and to fetal hypoxia.

DIFFERENTIAL DIAGNOSIS

The type of breech is diagnosed by ultrasound examination.

Immediate Management

- Prepare for Level 1 cesarean delivery in instances of footling breech or a premature breech fetus.
- If an epidural catheter is in place, it may be used for the delivery. If an epidural catheter is not in place, then general anesthesia is usually used.
- If the fetus is breech and if it is not footling or premature, management may differ. Vaginal delivery may be attempted. If vaginal delivery is attempted, both the anesthesiologist and the obstetrician must be prepared to perform a rapid cesarean delivery. Monitoring of the fetal heart tones must occur throughout the entire procedure.

Diagnostic Studies

Ultrasound is used to confirm physical examination.

Subsequent Management

Following delivery, the infant should be resuscitated by individuals skilled in neonatal resuscitation. Breech infants have more neonatal complications than neonates that have a vertex presentation, regardless of the route of delivery.

Risk Factors

- Maternal
 - Uterine anomalies
 - Multiparity
 - Pelvic tumors
- Fetal
 - Anencephaly
 - Microcephaly
 - Neuromuscular disorders
 - Polyhydramnios
 - Oligohydramnios
- Placental
 - Placenta previa

Prevention

Prior to delivery, the obstetrician may attempt an external version to turn the fetus from the breech presentation to cephalic presentation. This is a painful procedure, so epidural analgesia is beneficial. The use of epidural analgesia increases the success but does not increase the risk of abruption placenta from excessive pressure.

Special Considerations

- Before 28 weeks' gestation, approximately 40% of fetuses are in the breech position. At term, however, only 3%–4% of singleton pregnancies have a breech presentation. Due to the risks of breech vaginal delivery, the majority of breech presentations are delivered via elective cesarean sections. If a woman with a breech presentation begins to labor, the obstetrician will perform a cesarean section before the fetus becomes engaged. Breech delivery increases the risk of infection and perineal laceration for the mother. Footling breech may occur following rupture of the amniotic membranes. Footling breech is considered an obstetric emergency and will result in a Level 1 cesarean delivery. For other types of breech, the obstetrician will decide whether or not to proceed to cesarean delivery.
- There are circumstances in which a parturient and her obstetrician will decide to attempt a vaginal breech delivery, such as delivery of a nonvertex second twin, a multigravid parturient who has had several previous vaginal deliveries, or if the mother refuses a cesarean delivery. The Term Breech Trial randomized parturients with breech fetuses at 37 or more weeks' gestation to either planned cesarean delivery or planned breech vaginal delivery. There was an increased incidence of neonatal morbidity and mortality with attempted vaginal delivery. A breech vaginal delivery should only be considered for a nonviable fetus, delivery of a second nonvertex twin, or advanced labor with a breech presentation.

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Embolism: Thrombus, Amniotic Fluid, Air

Definition

Entry of solid (thrombus), liquid (amniotic fluid), or gas (air) into the maternal vascular system. Thromboembolism is the most common cause of maternal mortality and is also the most common form of embolism in pregnant patients.

Presentation

- Common to all:
 - Dyspnea
 - Chest pain
 - Tachypnea
 - Tachycardia
 - Increased central venous pressure
 - Dilated external jugular veins
 - Hypotension
- Amniotic fluid embolism includes all of the preceding and
 - Bleeding
 - Disseminated intravascular coagulopathy

Pathophysiology

After entry into the venous system, emboli lodge in the pulmonary vascular system, causing occlusion of the vessels and increased central venous blood pressure, decreased cardiac output, and hypotension. The resultant ventilation/perfusion mismatch causes hypoxemia. In contrast, amniotic fluid embolism is more consistent with an anaphylactoid reaction to one of the many possible mediators in amniotic fluid. The precise mechanism of the coagulopathy in amniotic fluid embolism is unknown but is postulated to be due to tissue factor, which is present in amniotic fluid, activating the extrinsic pathway by binding with Factor VII, activating Factor X, leading to clotting and a consumptive coagulopathy.

Immediate Management

- Thrombus
 - Increase FiO_2 to maintain oxygenation.
 - Consider endotracheal intubation if respiratory failure is imminent.
 - Provide hemodynamic support with fluids, inotropes, or vasopressors.

Immediate Management (continued)

- Anticoagulation with unfractionated heparin will be initiated by the obstetrician following resuscitation.
- In the setting of profound hemodynamic compromise, consider thrombolysis or surgery.
- Amniotic fluid
 - Increase FiO_2 to maintain oxygenation.
 - Consider endotracheal intubation if respiratory failure is imminent.
 - Provide hemodynamic support with fluids, inotropes, or vasopressors.
 - Consider insertion of an intra-arterial catheter and establishing central venous access.
 - The patient will develop a coagulopathy. Administer cryoprecipitate early to maintain an adequate fibrinogen level.
- Air
 - Inform the obstetrician and position the patient with the surgical site below the level of the heart. Placing the patient in reverse Trendelenburg position or returning the uterus into the abdomen will prevent the further entrainment of air.
 - Increase FiO_2 to maintain oxygenation. Intubation is usually not required.
 - Provide hemodynamic support with fluids, inotropes, or vasopressors.
 - Although a multi-orifice central venous catheter permits aspiration of air from the right atrium, the provider is usually too busy managing the hemodynamic consequences to attempt insertion.
 - The uterus should be wrapped in moist gauze to cover the open venous sinuses.
 - Consider hyperbaric oxygen postoperatively in severe cases.

DIFFERENTIAL DIAGNOSIS

- Asthma
- Anaphylaxis
- Sepsis
- Aspiration
- Peripartum cardiomyopathy
- Uterine rupture
- Placental abruption

- Hemorrhage
- Myocardial infarction

Diagnostic Studies

- Thrombus
 - Arterial blood gas analysis detects a decrease in PaO_2 and an increase in PaCO_2
 - V/Q scan
 - Spiral computed tomographic pulmonary angiography
 - Doppler ultrasound or magnetic resonance imaging to locate the source (usually the lower extremities)
- Amniotic fluid
 - The diagnosis of amniotic fluid embolism is primarily based upon clinical presentation and is one of exclusion. The diagnosis includes the presence of at least one of the following conditions: cardiac arrest, severe respiratory distress, seizure, or disseminated intravascular coagulopathy during pregnancy.
 - The aspiration of amniotic fluid debris from a catheter is not diagnostic for amniotic fluid embolism. The aspiration of squamous cells and lanugo hair has been aspirated from patients without amniotic fluid embolism.
 - Transesophageal echocardiography findings typically include a four-chamber view with right ventricular failure, suprasystemic right-sides pressures, bulging of the interatrial and interventricular septae, severe tricuspid regurgitation, and pericardial effusion.
- Air
 - Arterial blood gas analysis reveals a decrease in PaO_2 and an increase in PaCO_2 .
 - If general anesthesia is being used, there will be an abrupt decrease in end-tidal CO_2 .
 - Transesophageal and transthoracic echocardiography is both sensitive and specific. Transesophageal echocardiography requires general anesthesia for probe insertion.

Subsequent Management

- The most common cause of embolism in pregnancy is thrombus because pregnancy causes a hypercoagulable state with increased levels of Factors I, VII, VIII, X, and XII.
- Parturients with thromboembolism require anticoagulation. Prior to delivery, the patient is anticoagulated with low molecular weight heparin (warfarin is a teratogen). Follow guidelines for withholding low molecular weight heparin prior to attempting regional anesthesia or neuraxial labor analgesia.

- The hemodynamic and hematologic consequences of amniotic fluid embolism mandate that the patient be managed in the intensive care unit.
- Inhaled nitric oxide may be indicated for the pulmonary hypertension accompanying amniotic fluid embolism.
- If air embolism is suspected, take steps to stop vascular entrainment. Air enters the vascular circulation because of a pressure differential. Air entrainment occurs when the veins are stented open and the surgical site is above the level of the heart (negative venous pressure). Position the patient so that the surgical site is lower than the heart.

Risk Factors

- Thrombus: Age >35 years, obesity (BMI >30 kg/m²), cesarean delivery, current infection, parity >3, immobility, thrombophilia
- Amniotic fluid: Advanced maternal age, multiparity, tumultuous labor, trauma, multiple gestation, polyhydramnios, fetal macrosomia, augmentation of labor
- Air: Cesarean delivery (usually between delivery of infant to closure of hysterotomy), uterine exteriorization

Prevention

- Thrombus
 - Use of compression stockings during cesarean delivery
 - Mobility
 - SQ heparin
 - Antepartum pharmacologic thromboprophylaxis in patients with several risk factors or a history of thrombus
- Amniotic fluid
 - There is no proven means of prevention. Maintain a high index of suspicion.
 - Amniotic fluid embolism is a diagnosis of exclusion. Rule out other causes of hypotension and disseminated intravascular coagulation (DIC) before making the diagnosis of amniotic fluid embolism.
- Air
 - Position the surgical site below the level of the heart.

Special Considerations

- Embolism is the leading cause of maternal mortality in the United States. Air embolism is common during cesarean delivery, but typically does not result in clinical consequences.

Air can be demonstrated by transesophageal echocardiography in 93%–100% of cesarean deliveries. The most common time for air embolism is when the uterus is exteriorized to be repaired. At this point, the uterus and surgical incision are above the level of the heart, causing the entrainment of air. The pathophysiology of amniotic fluid embolism syndrome may be due to the immunologic reaction rather than to the embolism itself. Insulin-growth factor binding protein-1, which is a specific marker of amniotic fluid, is currently being evaluated. The use of recombinant Factor VIIa for the treatment of the bleeding accompanying amniotic fluid embolism is controversial. Although it decreases the amount of bleeding, it may also increase the risk of thromboembolism.

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Failed Intubation

Definition

Inability to intubate the trachea. The most common causes in the pregnant patient are pharyngeal, laryngeal, or tracheal edema.

Presentation

- Hypoxemia
- Difficulty with mask ventilation
- Difficulty with intubation
- Depending upon the degree of the hypoxemia, hypotension, hypertension, tachycardia, and bradycardia may occur.

Pathophysiology

During pregnancy, capillary engorgement of the mucosa throughout the respiratory tract causes swelling of the nasal and oral pharynx, larynx, and trachea, increasing the incidence of grade IV airways

by 34% from the first to the third trimester. Pregnant patients also become hypoxic more rapidly. The gravid uterus displaces the diaphragm 4–7 cm cephalad, decreasing functional residual capacity. Oxygen consumption is increased because of the developing fetus. Labor itself may also exacerbate an already difficult airway.

DIFFERENTIAL DIAGNOSIS

Failed intubation may occur during induction of general anesthesia or management complications such as oversedation or high spinal anesthesia. Loss of the airway may also occur at the end of general anesthesia if the patient is extubated prematurely.

Immediate Management

- If intubation is not possible
 - Call for help.
 - Attempt mask ventilation.
- If mask ventilation is possible, consider using cricoid pressure and mask ventilation for the cesarean delivery.
- If mask ventilation is not possible, follow the ASA Difficult Airway Algorithm.
- Consider using a laryngeal mask airway that allows for passage of a gastric tube.
- Consider inserting another type of supraglottic airway.
- Consider transtracheal jet ventilation.
- Consider a surgical airway (i.e., cricothyroidotomy).

Diagnostic Studies

- Mallampati classification and other features of the airway examination
 - Able to visualize
 - Faucial pillars, soft palate, uvula (Class 1)
 - Faucial pillars, soft palate (Class 2)
 - Soft palate only (Class 3)
 - Hard palate only (Class 4)
- The airway must be examined in every parturient *before* induction of anesthesia, regardless of whether the airway was examined previously.

Subsequent Management

Patients with difficult intubation should not be extubated until they have recovered sufficiently from anesthesia. Parturients who had received large quantities of intravenous fluids or blood products

may require postoperative intubation and mechanical ventilation until airway edema has resolved.

Risk Factors

- Obesity
- Previous airway surgery
- Diabetes mellitus
- Pre-eclampsia
- Inability to visualize oropharyngeal structures
- Receding mandible
- Short neck

Prevention

Be alert to the presence of risk factors that place the parturient at increased risk of complications from general anesthesia. In patients who have a suspected or known difficult airway, the obstetric team should encourage early initiation of epidural analgesia and the anesthesia team should ensure that the catheter is functional. Labor and delivery units should have equipment and the personnel readily available to manage airway emergencies. Basic airway equipment should be immediately available during the provision of regional anesthesia and include:

- Oxygen
- Suction
- Self-inflating bag and mask for positive pressure ventilation
- Laryngoscopes and assorted blades
- Videolaryngoscopy device
- Endotracheal tubes with stylets
- Medications for blood pressure support, muscle relaxation, and hypnosis.

In addition, portable equipment for difficult airway management should be readily available in the operative area of labor and delivery units to include:

- Rigid laryngoscope blades and handles of alternate design and shape
- Endotracheal tubes of assorted size
- Laryngeal mask airways of assorted sizes
- At least one device for emergency nonsurgical airway ventilation
 - Hollow jet ventilation stylet
 - Cricothyrotomy kit with or without a transtracheal jet ventilator
- Endotracheal tube guides
- Equipment suitable for emergency surgical airway access
- Topical anesthetics and vasoconstrictors

Special Considerations

- The most recently reported incidence of failed intubation in the obstetric population is 1:224 intubations. Appropriate training in emergency airway management will improve mortality from failed intubation.
- Pregnancy itself does not result in full stomach considerations. Pregnancy is associated with a shift in the position of the stomach that is caused by the gravid uterus. Although the angle of the gastroesophageal junction changes, this does not cause the pregnant patient to have a full stomach. If the pregnant patient has symptoms of a full stomach (e.g., frequent regurgitation, heartburn), she should be considered to be at risk for aspiration. All laboring patients should be considered a full stomach and at increased risk of aspiration. They should receive aspiration prophylaxis (sodium bicarbonate solution 30 mL and H₂ antagonists) prior to the induction of general anesthesia. If general anesthesia is required for a symptomatic pregnant patient or a patient in labor, a rapid sequence is indicated.
- Despite the advent of videolaryngoscopy, management of the difficult airway continues to be a problem in obstetric anesthesia. Videolaryngoscopy increases the chance of success with intubation but does not guarantee it.

Further Reading

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- Quinn AC, Milne D, Columb M, et al. Failed tracheal intubation in obstetric anaesthesia: 2 yr national case-control study in the UK. *Br J Anaesthesiol*. 2013; 110: 74–80.

Fetal Bradycardia

Definition

An abnormal baseline heart rate that is <110 beats per minute (bpm). Absence of baseline variability is highly predictive of abnormal fetal acid-base status. Currently, fetal heart rate (FHR) patterns are classified into one of three categories.

Category I (Figure 8.1):

- Baseline FHR 110–160 bpm
- Moderate FHR variability
- Lack of late or variable decelerations

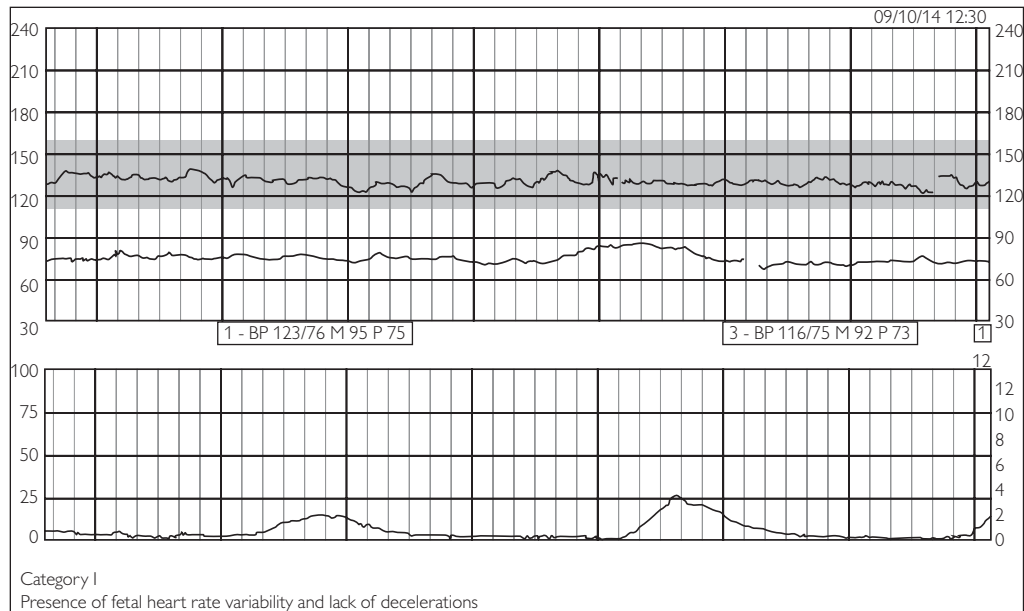


Figure 8.1 Category I. Presences of fetal heart rate variability and lack of decelerations.

- Early decelerations are present or absent
- Accelerations are present or absent

Category II (Figure 8.2):

- Fetal heart rate tracing not predictive of abnormal fetal acid-base status or indeterminate
- Not enough evidence to place in Category I or Category III
- Requires continued surveillance and re-evaluation
- Interpreted in the context of the clinical setting

Category III:

- Abnormal tracing
- Predictive of abnormal fetal acid-base status
- Absent baseline fetal heart rate variability (and any of the following):
 - Recurrent late decelerations
 - Recurrent variable decelerations
 - Bradycardia
- Sinusoidal pattern

Presentation

Clinical diagnosis is based on the FHR tracings.

Pathophysiology

Fetal bradycardia, when accompanied with decreased baseline variability or late decelerations, is associated with fetal acid-base abnormalities from fetal hypoxia. The etiology may be maternal (hypoxemia, hypotension, aortocaval compression, decreased hemoglobin), uterine (placental abruption, pre-eclampsia), or fetal (umbilical cord occlusion from knot or compression).

DIFFERENTIAL DIAGNOSIS

- Malpositioned sensor detecting maternal heart rate
- Maternal administration of beta-blockers
- False recording

Immediate Management

- Prepare for urgent cesarean delivery.
- Treat factors that may be contributing to fetal distress.
 - Administer supplemental oxygen.
 - Treat hypotension with fluid loading and incremental doses of phenylephrine (100 mcg) or ephedrine (ephedrine in large doses may decrease umbilical cord pH).
 - Left uterine displacement to treat aortocaval compression.

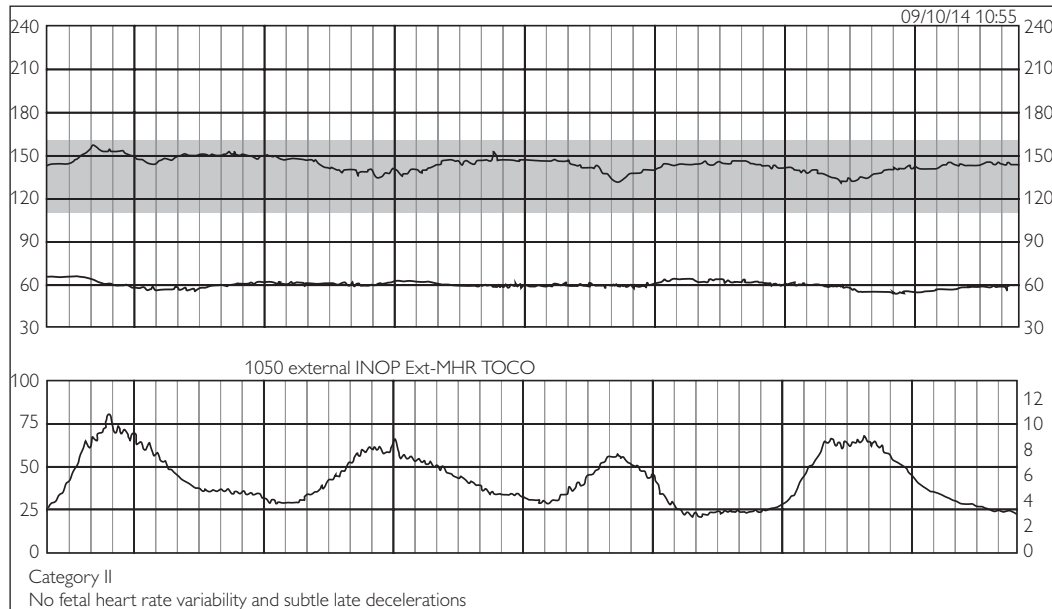


Figure 8.2 Category II. No fetal heart rate variability and subtle late decelerations.

Immediate Management (continued)

- If urgent delivery is required, consider aspiration prophylaxis with sodium bicarbonate.
- If a functioning epidural catheter is present, extend the sensory level with 3% 2-chloroprocaine or 2% lidocaine, depending upon the urgency of the case (2-chloroprocaine requires 2 minutes to achieve a satisfactory sensory level; lidocaine requires 4–6 minutes to achieve an appropriate sensory level).
- If no epidural catheter is in place, evaluate the maternal airway.
 - If the airway exam is suggestive of possible difficult intubation, consider regional anesthesia or videolaryngoscopy.
 - If airway exam does not suggest a possible difficult intubation, consider rapid sequence induction with cricoid pressure.
- If nonurgent delivery is planned, administer supplemental maternal oxygen and alter maternal position to prevent aortocaval obstruction.

Diagnostic Studies

The FHR may be confirmed with ultrasonography.

Subsequent Management

In case of urgent delivery, either general or regional anesthesia is administered, depending upon the amount of time available and the maternal airway examination. Communication between the obstetrician and anesthesiologist is critical in cases of fetal bradycardia. The obstetrician must convey the urgency of the delivery, and the anesthesiologist must convey his or her concerns.

Risk Factors

- Maternal hemorrhage
- Hypovolemia
- Maternal asthma
- Polyhydramnios
- Placental abruption
- Maternal cardiac disease
- Maternal cocaine use
- Maternal trauma
- Rupture of membranes with the fetal head not engaged
- Prematurity

Prevention

Maintaining uterine perfusion and fetal oxygen delivery decreases the risk of fetal bradycardia. Left uterine displacement helps to prevent aortocaval compression. Administer incremental doses of phenylephrine (100 mcg IV) or ephedrine (5 mg IV) to treat hypotension caused by neuraxial anesthesia. The use of supplemental oxygen is debatable because the impact on fetal oxygenation is minimal.

Special Considerations

- The baseline FHR is determined by approximating the FHR for a 10-minute window. Despite the presence of fetal bradycardia, the anesthesiologist may elect to perform regional anesthesia if a difficult airway is suspected in order to avoid failed intubation. Early initiation of epidural analgesia in patients with a Category II tracing is strongly recommended because it decreases the probability that general anesthesia will be required if the tracing progresses to Category III.

Further Reading

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Schnettler WT, Rogers J, Barber RE, Hacker MR. A modified fetal heart rate tracing interpretation system for prediction of cesarean section. *J Matern Fetal Neonatal Med*. 2012; 25: 1055–1058.

Hypotension

Definition

A systolic blood pressure <90 or 100 mm Hg or a decrease of 20% from its baseline.

Presentation

Maternal effects of hypotension are light headedness, dizziness, and nausea. The uterus does not autoregulate blood pressure, so hypotension that decreases uterine blood flow may result in a Category III FHR. A decrease in uterine blood also increases the risk of fetal acidosis.

Pathophysiology

Hypotension is caused by one of two mechanisms: a decrease in systemic vascular resistance or a decrease in cardiac output. In obstetric anesthesia, there are two main causes of hypotension: aortocaval compression and sympathectomy from neuraxial anesthesia. If the parturient lies on her back, the gravid uterus compresses the vena cava against the lumbar vertebra, decreasing venous return and cardiac output. This usually occurs after 20–24 weeks gestation. Aortocaval compression may be avoided by tilting the uterus to the left (uterine displacement) by placing a wedge beneath the right hip. Both epidural and spinal anesthesia can produce a sympathectomy, which decreases systemic vascular resistance. The uterus does not autoregulate blood flow, so blood flow is dependent upon the blood pressure.

Immediate Management

- Administer bolus doses of phenylephrine (100 mcg) or ephedrine (10 mg). Phenylephrine is preferred.
- Consider a starting phenylephrine infusion (50–100 mcg/min) for hypotension associated with spinal anesthesia.

DIFFERENTIAL DIAGNOSIS

- Aortocaval compression
- Sympathectomy from neuraxial anesthesia
- Anaphylaxis
- Sepsis
- Iatrogenic
- Concurrent medication
- Hemorrhage
- Hypovolemia
- Cardiac arrhythmia
- Pulmonary embolism
- Pneumothorax

Diagnostic Studies

- If aortocaval compression or neuraxial anesthesia are causing hypotension, no further studies are indicated.
- If another cause is suspected, serum hemoglobin or serum tryptase may be indicated.
- Additional studies include a blood gas, electrocardiogram, or chest X-ray, depending upon physical examination.

Subsequent Management

If the patient develops hypotension, maintain left uterine displacement and administer additional intravenous fluids. Determine the etiology of the hypotension.

Risk Factors

Beginning at 20–24 weeks gestation, the uterus becomes enlarged enough to result in aortocaval compression in the supine position (*supine hypotensive syndrome*). Many patients do not exhibit these symptoms when not anesthetized because the sympathetic nervous system is intact. However, with general or neuraxial anesthesia, the sympathetic nervous system is attenuated, making them more likely to develop supine hypotension.

Prevention

Originally, fluid loading with crystalloid solutions prior to neuraxial anesthesia for cesarean delivery was thought to decrease the incidence and severity of hypotension. However, fluid loading with these solutions does not prevent the development of hypotension. Colloid solutions have been demonstrated to decrease the incidence and severity of hypotension prior to neuraxial anesthesia. Colloid solutions are more expensive and have the risk of increased anaphylaxis. The routine administration of colloid prior to epidural or spinal anesthesia is not done. Prophylactic administration of vasopressors prior to neuraxial anesthesia is not recommended because they may cause hypertension. Most practitioners do not administer a fluid load prior to the initiation of epidural analgesia for labor.

Special Considerations

- The sympathetic nervous system runs from T1 to L2. The degree of hypotension is related to the degree of sympathetic block. The innervations for the first stage of labor is T10 to L1, making a high block unnecessary in the management of the pain of first stage. Limiting the degree of sympathectomy will decrease the risk of hypotension. For cesarean section, a T4 sensory level is required, causing a greater degree of hypotension as compared to analgesia for labor.
- Treatment with phenylephrine is associated with a higher umbilical cord blood pH than is ephedrine. Therefore, phenylephrine is preferred for the management of hypotension accompanying spinal anesthesia. This difference is not due to an effect on uterine blood flow. Ephedrine crosses the placenta and stimulates the fetal sympathetic nervous system when administered in large quantities.

- Many practitioners initiate a continuous infusion of phenylephrine for the treatment of hypotension accompanying spinal anesthesia during cesarean section. A continuous infusion of phenylephrine decreases the incidence of maternal nausea/vomiting as compared to intermittent boluses.

Further Reading

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Habib AS. A review of the impact of phenylephrine administration on maternal hemodynamics and maternal and neonatal outcomes in women undergoing cesarean delivery under spinal anesthesia. *Anesth Analg.* 2012; 114: 377–390.

Local Anesthetic Toxicity

Definition

Unintended systemic effects of local anesthetics usually due to unintended intravascular injection; also may occur from systemic absorption of excessive amounts.

Presentation

The symptoms from local anesthetic occur when various concentrations are achieved in the blood. Toxicity may present with central nervous system and cardiovascular symptoms. Typically, central nervous system effects occur before cardiovascular effects.

- Low blood concentrations: The patient may note tongue numbness, metallic taste, or tinnitus.
- High blood concentrations: As the concentrations increase, the patient will seize before becoming comatose.
- Very high blood concentrations: If the concentrations of the local anesthetic continue to increase, the patient will develop cardiovascular symptoms in the form of myocardial depression, dysrhythmias, and cardiovascular collapse. These effects occur because of the local anesthetic impairing electrical conduction in the myocardium with ventricular tachycardia and ventricular fibrillation, and myocardial depression.

Pathophysiology

Local anesthetics cause reversible blockade of the sodium channels. They act on the central and peripheral nervous system, myocardial muscle, and the conduction system in the myocardium. Seizures are caused by the local anesthetic blocking inhibitory

neurons within the central nervous system, resulting in unopposed excitatory impulses. Eventually, these too are blocked, resulting in coma. Cardiovascular toxicity results from blockade of the sodium channels in the muscle and conduction system of the heart, leading to myocardial depression and dysrhythmias.

Immediate Management

- Halt the injection of local anesthetic.
- Prepare for maternal cardiac arrest and initiation of cardiopulmonary resuscitation.
- If the patient is seizing, administer either a benzodiazepine (e.g., midazolam 2 mg IV) or propofol 1–2 mg/kg. Prepare to secure the airway.
- Check for a maternal pulse. If there is no pulse, begin cardiopulmonary resuscitation and prepare for cesarean delivery (see section on maternal cardiac arrest).
- Administer 20% intralipid 1.5 mL/kg IV bolus followed by 0.25 mL/kg/min for 30–60 minutes.
- Additional boluses of 20% intralipid may be indicated if the symptoms persist.

DIFFERENTIAL DIAGNOSIS

- Eclampsia
- Underlying seizure disorder
- Toxic from other drugs
- Metabolic from electrolyte or glucose abnormalities
- Alcohol/drug withdrawal
- Pulmonary embolism
- Cardiac arrest
- Myocardial infarction
- Underlying cardiac disease

Diagnostic Studies

There are no diagnostic studies for local anesthetic toxicity. If local anesthetic toxicity is suspected, the administration of intravenous intralipid should not be delayed.

Subsequent Management

- If symptoms persist, administer a second dose of intralipid.
- If the patient's symptoms do not respond to intralipid, another etiology of the seizure or myocardial arrhythmia should be considered.

- Following resolution of the symptoms, a neurologic examination should be performed. If the patient had a cardiac arrhythmia, the patient should have continuous cardiac monitoring as well as laboratory testing for myocardial infarction.

Risk Factors

Parturients are at risk for intravascular placement of epidural catheters due to dilation of the epidural veins.

Prevention

Prior to the administration of local anesthetic for cesarean delivery, a test dose of 3 mL 1.5% lidocaine with epinephrine 1:200,000 should be administered. If the catheter is intravascular, there will be an increase in maternal heart rate of 10–15 bpm. It is common for the maternal heart rate to vary during labor due to the pain of contractions. If a patient should experience a contraction during the administration of the test dose and the heart rate increases, this increase may be a “false positive,” meaning that the increase was because of pain, not because of intravascular injection.

Fractionate the dose of local anesthetic and ask the patient about tinnitus and check for tachycardia during injection. Subsequent doses of epidural anesthesia should be fractionated. Administer 3–5 mL increments, waiting several minutes between doses. An intravascular injection may be detected prior to giving the full dose of local anesthetic.

Special Considerations

- Any location in which local anesthetics are administered should have the 20% intralipid in a sufficient quantity to treat toxicity readily available. There should also be a card with instructions for its use.

Further Reading

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Neal JM, Mulroy MF, Weinberg GL. American Society of Regional Anesthesia and Pain Medicine checklist for managing local anesthetic systemic toxicity: 2012 version. *Reg Anesth Pain Med.* 2012; 37: 16–18.

Maternal Cardiac Arrest

Definition

Absence of a palpable maternal pulse requiring cardiopulmonary resuscitation.

Presentation

The mother has no palpable pulse. The electrocardiogram will reveal pulseless electrical activity, ventricular fibrillation, or asystole.

Pathophysiology

The most common cause is venous thromboembolism. The American Heart Association uses the mnemonic to assist with the diagnosis of the etiology of maternal cardiac arrest: BEAUCHOPS. The letters correspond to the following etiologies: B—bleeding; E—embolism (venous and amniotic fluid); A—anesthetic complications; U—uterine atony (most likely leading to bleeding); C—cardiac disease (myocardial infarction, pre-existing cardiac disease, cardiomyopathy); H—hypertension (pre-eclampsia and eclampsia, intracerebral bleed); O—other (differential diagnosis of standard ACLS guidelines); P—placental abruption/previa (bleeding and possible disseminated intravascular coagulation); and S—sepsis. The anatomic and physiologic changes of pregnancy and labor result in significantly decreased cardiovascular and pulmonary reserves, which may complicate the resuscitation, and both the mother and fetus must be considered.

DIFFERENTIAL DIAGNOSIS

- Hemorrhage
- Total spinal anesthesia
- Local anesthetic toxicity
- Anaphylaxis
- Embolism
- Maternal cardiac disease
- Sepsis
- Eclampsia
- Intracerebral hemorrhage

Immediate Management

- The goal of resuscitating the parturient is the return of maternal circulation. Follow the American Heart Association ACLS algorithm.
- Begin chest compressions immediately.

Immediate Management (*continued*)

- Left uterine displacement should be used to relieve aortocaval compression. Left uterine displacement is best achieved manually rather than tilting the bed. Tilting the bed or a wedge may decrease the effectiveness of chest compressions.
- For chest compressions, the hands may need to be placed higher on the sternum than usual due to the gravid uterus.
- Ventilate the patient. Intubation of the pregnant patient may be more difficult. Use capnography to document adequate ventilation and chest compressions.
- If indicated, defibrillate the patient. The energy required for defibrillation does not change during pregnancy. The energy settings recommended in the ACLS algorithm apply for pregnant patients.
- Medications should be administered in the same doses as would be used for a nonpregnant patient. Although the physiologic changes of pregnancy may alter the volume of distribution of the various medications, no adjustment is required.
- If local anesthetic toxicity is suspected, intravenous intralipid is the indicated treatment.
- Use the “5-minute rule” during resuscitation of the pregnant patient. If the maternal circulation has not returned by 4 minutes of cardiopulmonary resuscitation, cesarean delivery should be performed with delivery of the infant by minute 5.

Diagnostic Studies

An automated external defibrillator will determine whether the cardiac rhythm is treatable with electricity. The etiology of pulseless electrical activity should be investigated with additional laboratory information such as arterial blood gas analysis and transthoracic echocardiography.

Subsequent Management

If the maternal circulation has not been restored by 4 minutes, the obstetrician should perform cesarean delivery with the goal of delivery of the baby by minute 5. Performance of cardiopulmonary resuscitation in a pregnant patient is difficult due to the gravid uterus causing vena cava compression and inhibiting effective chest compressions.

Risk Factors

Parturients with congenital heart disease are at particular risk for cardiac arrest during pregnancy. Pregnancy is associated with an increase in coagulation factors, including fibrinogen (Factor I), proconvertin (Factor VII), antihemophilic factor (Factor VIII), Christmas factor (Factor IX), Stuart-Prower factor (Factor X), and Hageman factor (Factor XII). The concentrations of Factors I and VIII increase by >100%. Parturients are more likely to be hypercoagulable and at risk for thromboembolism because factor concentrations are increased.

Prevention

Appropriate testing of the epidural catheter may reduce the incidence of total spinal anesthesia and local anesthetic toxicity. Due to aortocaval compression by the gravid uterus, the epidural veins are dilated and are easily punctured during epidural catheter placement. Dosing in an epidural catheter should always be in a divided fashion, eliciting symptoms from the patient. Also, a high level of suspicion in parturients with cardiac disease, hemorrhage, or hypertensive disorders of pregnancy should be maintained throughout the peripartum period.

Special Considerations

- The incidence of cardiac arrest in the pregnant patient is estimated to be <1:20,000 women. The most important step in the resuscitation of the parturient is the early delivery of the fetus if there is no return of maternal circulation. Effective cardiopulmonary resuscitation in the pregnant patient is difficult because chest compressions are less effective and the gravid uterus obstructs the vena cava. Early delivery decreases the risk of neurologic injury in the infant and may improve the likelihood of successful maternal resuscitation by decreasing aortocaval compression and improving maternal compression. Estimated gestational age is an important factor in predicting prognosis for infants after perimortem cesarean deliveries. The threshold for expected fetal viability is considered to be around 24 weeks' gestation. Although emergency delivery performed between 20 and 23 weeks may produce a nonviable infant, it may enable successful resuscitation of the mother.

Further Reading

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Mhyre JM, Tsen LC, Einav S, et al. Cardiac arrest during hospitalization for delivery in the United States, 1998–2011. *Anesthesiology*. 2014; 120: 810–818.

Maternal Hemorrhage

Definition

The definition of hemorrhage is difficult because the parturient undergoes several physiologic changes of pregnancy, which result in an increase in blood volume to compensate for the blood loss during delivery. Most physicians use as a definition a blood loss of 500 mL for a vaginal delivery and 1000 for a cesarean delivery as a definition, but the majority of deliveries have this amount of blood loss. Symptoms such as hypotension or oliguria do not occur until a blood loss >10% blood volume.

Presentation

Parturients with obstetric hemorrhage present with:

- Increased maternal heart rate (sinus tachycardia)
- Maternal hypotension
- Fetal tachycardia

Pathophysiology

At term, uterine blood flow is approximately 500–700 mL/min. Hemorrhage during delivery may be either antepartum or postpartum. The two major causes of antepartum hemorrhage are placenta previa and placental abruption. The major causes of postpartum hemorrhage are uterine atony, retained placenta, and placenta accreta. Placenta previa occurs when the placenta overlies the cervical os, or is proximate to the internal os of the cervix. Placental abruption refers to separation of the placenta after 20 weeks gestation but before the birth of the fetus. Placenta accreta is defined as implantation directly onto the myometrium (accreta), into the myometrium (incretta), or through the myometrium (percreta). If the placenta is implanted into the myometrium, the uterus cannot contract, causing hemorrhage. The incidence of postpartum hemorrhage is increasing; the etiology is unclear. A soft, poorly contracted uterus is referred to as *uterine atony*.

DIFFERENTIAL DIAGNOSIS

- Antepartum hemorrhage: Placenta previa results in painless vaginal bleeding with no concealed bleeding. Placental abruption is very painful, but the blood loss may be retroplacental and concealed.

- Postpartum hemorrhage: The incidence of postpartum hemorrhage is increasing. Many attribute it to the increase in labor induction, making the uterus resistant to oxytocin.

Immediate Management

- Manage the hemorrhage:
 - Obtain large-bore intravenous access.
 - Initiate fluid resuscitation.
 - Initiate rapid transfusion protocol.
 - Every labor floor should have a rapid transfusion protocol that will allow the provider to access packed red blood cells, fresh-frozen plasma, and platelets. As most cases of obstetric hemorrhage are accompanied by a low fibrinogen, the use of cryoprecipitate should be used early in the resuscitation.
 - Use cross-matched blood if possible. If the blood type is unknown, O-blood should be used.
- Management of uterine atony:
 - Uterine atony results from the lack of uterine contraction. The obstetrician will initially attempt manual massage (fundal massage) to get the uterus to contract. If that is unsuccessful, the choice of medication depends upon the practitioner as there is no study demonstrating the superiority of one medication over another.
 - Oxytocin, 20–40 U, is added to 1 L crystalloid solution and this solution is administered intravenously. Rapid administration may cause hypotension.
 - Methylergonovine 0.2 mg IM. Increased blood pressure is a relative contraindication.
 - Prostaglandin $F_{2\alpha}$ 0.25 mg IM. Asthma is a contraindication.
- Management of placenta accreta:
 - Prepare for significant hemorrhage: Establish large-bore IV access.
 - Significant hemorrhage may occur during the hysterectomy. Fluid replacement should consist of packed red blood cells, fresh-frozen plasma, and platelets in a 1:1:1 fashion. Strong consideration should be given to the administration of cryoprecipitate (hypofibrinogenemia). Recombinant Factor VII decreases bleeding but may increase the risk of thromboembolism.
 - Consider general anesthesia. Although a gravid hysterectomy may be performed under regional anesthesia, the provider will be transfusing blood products and may have difficulty managing the airway if this becomes necessary.

Diagnostic Studies

- Placenta previa is diagnosed via ultrasound. If the patient has a placenta previa, a cervical exam is *not* performed due to risk of causing bleeding.
- Placental abruption and retained placenta are diagnosed with ultrasound.
- Uterine atony is a diagnosis of exclusion.

Subsequent Management

- If the patient has a placenta previa, cesarean delivery is necessary. For placenta previa, the patient will bleed only when the cervix begins to dilate. As such, the patient will not bleed if not in labor. The goal for the obstetrician is to allow the fetus to mature in utero as long as possible, but not so long as to allow the mother to begin to labor and to hemorrhage.
- If the patient has a placenta abruption, delivery may be by vaginal or cesarean delivery. Placental abruption is the leading cause of disseminated intravascular coagulation. Therefore, coagulation studies and platelet count should be followed if placental abruption is suspected.
- The management of uterine atony will depend upon subsequent events. If the uterus begins to contract after a period of atony, no further intervention is required. Oxytocin may be continued to maintain uterine tone. If retained products are suspected, intravenous nitroglycerin provides uterine relaxation and allows the obstetrician to explore the uterus manually, potentially avoiding dilation and evacuation.
 - The obstetrician will request additional medications such as methylergonovine or prostaglandin $F_{2\alpha}$.
 - For continued bleeding without hemodynamic instability, the obstetrician may request an interventional radiology consultation for arterial embolization.

Risk Factors

- Placenta previa
 - Previous cesarean delivery
 - Previous uterine surgery
 - Older maternal age
 - Multiple pregnancy
- Placental abruption
 - Pre-eclampsia
 - Hypertension
 - History of placental abruption

Risk Factors (continued)

- Cocaine
- Trauma
- Placenta accreta
 - Previous cesarean section and placenta previa
- Uterine atony
 - Multiparity
 - Multiple gestation
 - Infection
 - Magnesium
 - Fetal macrosomia
 - Polyhydramnios

Prevention

There are no specific means to prevent obstetric hemorrhage, but early identification of risk factors and preparation for extensive blood loss are essentials. Establish large-bore IV access in patients who are at risk, and verify that blood products are immediately available. In patients with placenta accreta, consider an interventional radiology consultation prior to cesarean delivery to insert balloon catheters in the iliac arteries. These catheters may be inflated after delivery to decrease uterine blood flow.

Special Considerations

- The use of cell saver in obstetrics is controversial because of the theoretical risk of infection and amniotic fluid embolism. Both the American Congress of Obstetricians and Gynecologists and the American Society of Anesthesiologists guidelines state that the use of a cell saver should be considered if available. Recombinant Factor VIIa has been used, but may exacerbate a pre-existing hypercoagulable state.

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Neonatal Resuscitation

Definition

During the transition from intrauterine to extrauterine life, the neonate is required to make rapid and profound physiologic changes as the neonate transfers from receiving oxygen from uterine blood flow and nonfunctioning lungs to receiving oxygen completely from respiration. Approximately 10% of newborns require some assistance to initiate respiration, whereas about 1% of newborns need extensive resuscitative measures

Presentation

- Failure to initiate spontaneous ventilation after being stimulated (usually by gentle rubbing and suctioning)
- Heart rate <100 bpm

Pathophysiology

Before birth, the fetus depends completely on uterine blood flow for delivery of oxygen to the developing tissues. The transition to postnatal circulation, fluid in the alveoli must be absorbed into the lung tissue and replaced by air. The umbilical arteries and vein are clamped, removing the low-resistance placental circuit and increasing systemic blood pressure. Vasodilation decreases pulmonary artery resistance, resulting in increased pulmonary blood flow and decreased flow through the ductus arteriosus, which begins to constrict. At the completion of this transition, the baby is breathing spontaneously and maintaining the oxygen saturation on room air. If this sequence is interrupted, the pulmonary arterioles may remain constricted and the systemic arterial blood will not become oxygenated.

DIFFERENTIAL DIAGNOSIS

- Maternal opioid consumption
- Other causes of apnea include general anesthesia administered to the mother, congenital heart defects or other malformations, and neonatal sepsis.

Immediate Management

- Place the infant on a radiant warmer to prevent hypothermia.
- Dry and stimulate the infant for 20 seconds.
- If the infant remains apneic following stimulation, assume that the neonate is experiencing secondary apnea and requires positive pressure ventilation.

Immediate Management (continued)

- Attach a pulse oximeter.
- Ventilation should be with room air for term neonates and with a mixture of room air/oxygen for premature infants. If the neonate requires chest compression, ventilation should be with 100% oxygen.
- Administer intravenous naloxone, 0.1 mg/kg, if maternal opioid consumption is suspected.
- Initiate positive pressure ventilation at a rate of 40–60 breaths per minute at a pressure <20 cm H₂O (a pressure manometer must be used during neonatal resuscitation).
- After initiation of ventilation, assess the heart rate by auscultation or electrocardiogram (preferred). (An increasing heart rate is the most reliable indication of effective ventilation.)
 - If the heart rate is <100 bpm, assess adequacy of ventilation and make necessary adjustments.
 - If the heart rate is <60 bpm, initiate chest compressions. Chest compressions may be performed by having the hand encircle the chest with the two thumbs performing the compressions or by two fingers compressing the lower sternum. The two-thumb technique is preferred because it generates greater systolic and coronary perfusion pressures.
 - The rate of compression is one breath after every third compression, for a total of 30 breaths and 90 compressions per minute.
- If the heart rate is >60 bpm, chest compressions are stopped but ventilation is continued.
- If the heart rate is >100 bpm and the baby begins to breathe spontaneously, positive ventilation is stopped.
- If the heart rate remains <60 bpm despite effective ventilation, epinephrine 1:10,000 0.1–0.3 mL/kg is administered intravenously.

Subsequent Management

If the infant responds to drying or to positive pressure ventilation, no further workup is necessary. After resuscitation, the infant should be admitted to the neonatal intensive care unit for further management.

It is permissible to withhold resuscitation if the infant's age, weight, or coexisting condition is associated with a high mortality. If the infant does not have an established heart rate following 10 minutes of resuscitation, it is permissible to discontinue resuscitative

efforts. In both of these situations, a consistent and coordinated approach that considers the available medical literature must be followed.

Risk Factors

- Maternal diabetes
- Pre-eclampsia
- Maternal infection
- Maternal drug overdose
- Post-term gestation
- Fetal malformation
- Meconium
- Nonreassuring fetal heart tracing during delivery
- Emergency cesarean delivery
- Prolonged rupture of membrane
- Premature labor
- Instrumental delivery (forceps or vacuum)

Prevention

If the need for neonatal resuscitation is anticipated because of the presence of a risk factor, equipment and personnel should be immediately available at delivery. The absence of a risk factor does not guarantee that the infant will not require resuscitation. Individuals capable of providing neonatal resuscitation should be available in every delivery unit.

Special Considerations

- Respirations are the first vital sign to be affected when a newborn is deprived of oxygen. After an initial period of attempts to breathe, there is a period of primary apnea. During primary apnea, stimulation, such as drying the infant, will cause a resumption of breathing. If hypoxia continues, the baby gasps and enters a period of secondary apnea. During secondary apnea, stimulation will not restart respiratory efforts. The only therapeutic for secondary apnea is positive pressure ventilation. At least one person (not the anesthesia provider) whose primary responsibility is the neonate and who is capable of initiating resuscitation should be present at every delivery.
- Laryngeal mask airways have proven effective in the management of ventilation of infants >2 kg or 34 weeks gestation. The use of the laryngeal mask airway for smaller or younger infants has limited data.

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Pre-eclampsia

Definition

Pre-eclampsia is a multisystem disorder associated with increased blood pressure, proteinuria, and edema. Blood pressure must be ≥ 140 mm Hg systolic or ≥ 90 mm Hg diastolic on two measurements that are at least 4 hours apart. Hypertension must be accompanied by proteinuria (as defined as ≥ 300 mg/24 hours or protein/creatinine ratio >0.3) or in the absence of proteinuria, have any of the following: thrombocytopenia, renal insufficiency (creatinine >1.1 mg/dL), impaired liver function, pulmonary edema, or cerebral or visual symptoms. Severe pre-eclampsia occurs with one of the following symptoms: systolic blood pressure of >160 mm Hg or diastolic blood pressure of >110 mm Hg, thrombocytopenia, impaired liver function, progressive renal insufficiency, pulmonary edema, or new-onset cerebral or visual disturbances. Eclampsia includes these findings as well as central nervous system involvement leading to seizures not from other cerebral conditions.

Presentation

Pre-eclampsia

- Systolic blood pressure >140 mm Hg
- Diastolic blood pressure >90 mm Hg
- Proteinuria >300 mg/24 hours
- If no proteinuria, may still diagnose pre-eclampsia if any of the following symptoms exist:
 - Platelet count $<100,000/\mu\text{L}$
 - Liver transaminases twice normal concentration
 - Creatinine >1.1 mg/dL
 - Pulmonary edema
 - New-onset cerebral or visual disturbance

Severe pre-eclampsia

- Increased blood pressure and any of the following:
 - Systolic blood pressure >160 mm Hg
 - Diastolic blood pressure >110 mm Hg
 - Platelet count $<100,000/\mu\text{L}$
 - Right upper quadrant pain and elevated liver enzymes
 - Serum creatinine >1.1 mg/dL
 - Pulmonary edema
 - New-onset cerebral or visual disturbances

Pathophysiology

Hypertension during pregnancy has four potential causes: 1) pre-eclampsia-eclampsia, 2) chronic hypertension, 3) chronic hypertension with superimposed pre-eclampsia, and 4) gestational hypertension. The etiology of pre-eclampsia remains unknown, but the disease most likely begins at implantation, well before clinical symptoms appear. The placenta is the most likely source of the disease because molar and abdominal pregnancies have also been associated with pre-eclampsia. The leading theory involves an immunologic alteration of trophoblast function and decreased vascularity. The decrease in vasculature leads to ischemia and the generation of free radicals, which causes the symptoms. There is an abnormal response to the angiogenic proteins produced by the placenta, resulting in the abnormal vasculature.

Immediate Management

- Pre-eclampsia after 36 weeks gestation or the presence of severe pre-eclampsia is an indication for delivery either by induction of labor or cesarean section.
- Intravenous magnesium is administered to women with pre-eclampsia to prevent the progression of the disease to eclampsia (seizures). As it is not possible to determine who will develop eclampsia, all women with pre-eclampsia should receive magnesium. While other anticonvulsants have been studied, none has been as effective as magnesium.
- Consider epidural labor analgesia or neuraxial anesthesia (including spinal anesthesia) for cesarean delivery, even if the patient has severe pre-eclampsia.
- Check the platelet count in all patients before attempting regional anesthesia.

DIFFERENTIAL DIAGNOSIS

- Local anesthetic toxicity
- Gestational hypertension
- Hypertension
- Acute cocaine toxicity
- Pain
- Pre-existing kidney disease
- Toxic/metabolic
- Alcohol/drug withdrawal

Diagnostic Studies

- Proteinuria is diagnosed with a timed urine collection. Proteinuria up to 300 mg/24 hours is normal. Proteinuria >300 mg/24 hours is abnormal. Using a dipstick of the urine for protein is too nonspecific for the diagnosis. A protein/creatinine ratio of at least 0.3 is also consistent with pre-eclampsia, but it is possible for a patient to have pre-eclampsia without proteinuria.
- Complete blood count including the platelet count.
- The hemoglobin level may be increased due to hypovolemia or it may be decreased if hemolysis is occurring.
- If a patient seizes, a head computed tomography scan will rule out an anatomic etiology.
- Although placental growth factor and sFlt-1 may be measured in the blood or the urine, their variability makes them unsuitable for the diagnosis or for the prediction of the disease at this point. It is unclear of the role of these blood tests in the future.

Subsequent Management

- Magnesium sulfate is started for seizure prophylaxis in all pre-eclamptic patients. In patients with normal renal function, use a loading dose of 4 grams intravenously followed by an infusion of 1–2 g/h.
- The diagnosis of severe pre-eclampsia indicates that the mother is at risk of end-organ damage and is an indication for delivery.
- Parturients with preeclampsia are at risk for the development of eclampsia in the postpartum period, especially during the first 24 hours. Patients must be monitored and magnesium should be continued for 24 hours after delivery.
- If a patient develops thrombocytopenia after an epidural catheter has been inserted, removal should be delayed until the

platelet count begins to normalize, which may take up to 3 days postpartum.

Risk Factors

- First pregnancy
- Previous pre-eclampsia
- Chronic hypertension or chronic renal disease
- History of thrombophilia
- Multigestation pregnancy
- Family history of pre-eclampsia
- Diabetes mellitus
- Obesity
- Systemic lupus erythematosus
- Advanced maternal age (>40 years)

Prevention

There is no known intervention that prevents pre-eclampsia. For women with a history of early onset pre-eclampsia and a preterm delivery in more than one prior pregnancy, the daily administration of aspirin 60–80 mg is indicated, starting in the first trimester.

Special Considerations

- The incidence of pre-eclampsia in the United States is increasing and has increased by 25% over the past 20 years. It is the leading cause of maternal and perinatal morbidity and mortality, and is a risk factor for future cardiovascular disease. It complicates up to 10% of pregnancies and is more likely to occur at both extremes of reproductive age.
- One percent of parturients with pre-eclampsia will develop eclampsia.
- Spinal anesthesia is not contraindicated in patients with severe pre-eclampsia, nor is it associated with a greater degree of hypotension or pulmonary edema.
- Thrombocytopenia is the most common hematologic abnormality in patients with pre-eclampsia. Its incidence depends upon the severity of the disease and the presence of placental abruption. The platelet count should be checked prior to the initiation of neuraxial anesthesia. The American Society of Anesthesiologists has not recommended a safe limit for the platelet count in patients with pre-eclampsia, and there are numerous case reports of epidural placement in patients with low platelet counts.

- Magnesium sulfate significantly potentiates nondepolarizing neuromuscular blocking drugs. If the patient receives general anesthesia for cesarean delivery, nondepolarizing neuromuscular blocking drugs should be avoided if possible.

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Sepsis

Definition

Puerperal sepsis is an infection of the genital tract occurring at any time between rupture of the membranes and 42 days postpartum.

Presentation

The patient should have two or more of the following: pelvic pain, fever $>38.5^{\circ}\text{C}$, abnormal vaginal discharge, and delay in the reduction of the size of the uterus. Systemic inflammatory response syndrome (SIRS) is an inflammatory process with two or more of the following clinical findings are present: temperature $>38.5^{\circ}\text{C}$, heart rate >90 bpm, respiratory rate >20 /min, and white blood cell count $>12 \times 10^9$ /dL. Sepsis is SIRS with an infection. In septic shock, the patient is hypotensive.

Pathophysiology

The majority of cases of sepsis during pregnancy involved Group A streptococcus. Approximately 5%–30% of the population is thought to be asymptomatic carriers. Group B streptococcus may cause urosepsis or endometritis with approximately 20%–30% of women of reproductive age having these bacteria in the vagina. Influenza A and B are the most common pathogens of pneumonia in pregnancy.

Immediate Management

- Draw blood for culture and blood count.
- Obtain uterine swabs for culture.
- Begin high dose broad spectrum antibiotics. Note: Blood cultures should be obtained before initiation of antibiotics.
- Septic shock is treated with vasopressors such as phenylephrine and intravenous fluids.
- Antibiotics should be continued for 7–10 days.

DIFFERENTIAL DIAGNOSIS

- Asthma
- Anaphylaxis
- Hypovolemia
- Hemorrhage
- Thromboembolism
- Pneumothorax
- Pericardial effusion

Diagnostic Studies

- Obtain cultures, including blood, vaginal swabs, surgical sites, and urine.
- Draw blood for blood count, electrolytes, C-reactive protein, and lactate.
- Obtain an arterial blood gas analysis if pneumonia is suspected.
- Radiographic studies of the chest or abdomen are guided by the patient's symptoms.

Subsequent Management

Continue treatment with intravenous fluids. Both colloids and crystalloids have been used for the fluid resuscitation. If the patient remains hypotensive, initiate treatment with one or more vasopressors as required (e.g., norepinephrine, epinephrine, or phenylephrine). Follow serum lactates to determine the response to therapy. Broad-spectrum antibiotics should be initiated.

Risk Factors

- History of group B streptococcal infection
- Vaginal discharge
- History of pelvic infection
- Prolonged rupture of membranes

Risk Factors (continued)

- Cesarean delivery
- Recent upper respiratory infection
- Diabetes
- Human immunodeficiency virus
- Maternal age >35 years
- Low socioeconomic factors

Prevention

Preoperative skin preparation prior to cesarean delivery should include hair removal with an electric razor and antiseptic (chlorhexidine). All patients having cesarean delivery should receive prophylactic antibiotics. A surgical mask and gown should be worn by all personnel during both vaginal delivery and cesarean delivery. Hand hygiene should be enforced throughout the labor suite.

Special Considerations

- Sepsis remains a major cause of maternal mortality. The physiologic changes of pregnancy result in an increase in maternal heart rate and respiratory rate, making the diagnosis of SIRS difficult. Pregnancy and labor also increase the white blood cell count in some individuals. A low threshold for the diagnosis of sepsis should be maintained given the associated high mortality.

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Shoulder Dystocia**Definition**

Shoulder dystocia occurs when gentle downward traction on the fetal head by the obstetrician fails to result in delivery of the shoulders. The most common reason is impaction of the anterior shoulder under the pubic bone. Less commonly, the posterior shoulder becomes impacted on the sacral promontory.

Presentation

- Prolonged second stage of labor
- Following delivery of the fetal head, gentle traction on the head fails to result in delivery of the shoulder.
- The obstetrician does not apply excessive force to the fetal head to prevent brachial plexus injury.

Pathophysiology

During a normal delivery, the fetal head and shoulders rotate to allow for its descent and passage through the maternal pelvis. If the shoulder should rotate into the anterior-posterior diameter before entering the pelvis, the shoulders may become impacted on the pelvic bone. Another concern with shoulder dystocia is compression of the umbilical cord against the maternal pelvis with no flow of oxygenated blood to the fetus.

DIFFERENTIAL DIAGNOSIS

- Poor maternal pushing effort
- Absence of maternal contraction

233

Immediate Management

- Delivery must be expedited to prevent hypoxic brain injury.
- Prepare for emergency cesarean delivery.
- To assist with the vaginal delivery, the obstetrician will perform the *McRoberts maneuver*, in which the maternal legs are hyperflexed to the maternal abdomen. This increases the size of the maternal pelvis by flattening of the maternal lumbar lordosis and cephalad rotation of the symphysis. Another individual will apply suprapubic pressure to decrease the diameter of the shoulders. If attempts to deliver the neonate fail, the obstetrician may return the fetal head into the maternal pelvis and perform cesarean delivery (the *Zavenelli maneuver*). Both require maternal analgesia.

Diagnostic Studies

Clinical diagnosis is made by the obstetrician.

Subsequent Management

Following delivery, the infant should be evaluated by a neonatal resuscitation team. The mother should be informed of the event with the medical record documenting the times and the maneuvers used.

Risk Factors

- Fetal macrosomia
- Abnormal maternal pelvic anatomy
- Gestational diabetes
- Postdates pregnancy
- Maternal obesity
- Prolonged second stage
- Previous shoulder dystocia

Prevention

Assess fetal position during labor and delivery. The obstetrician will strongly consider elective cesarean delivery in patients at risk for shoulder dystocia. Simulation training improves the management of shoulder dystocia by improving the functioning of a multidisciplinary team.

Special Considerations

- Shoulder dystocia complicates an estimated 0.6%–1.4% of all vaginal deliveries and requires an immediate and coordinated response. The anesthesia provider must be prepared to provide analgesia if an epidural catheter is present and to provide anesthesia if urgent cesarean delivery is required.
- Potential maternal complications include:
 - Hemorrhage
 - 4° vaginal laceration with the potential for the development of a rectovaginal fistula
 - Pubic symphyseal separation
 - Uterine rupture
- Potential neonatal complications include:
 - Hypoxic encephalopathy
 - Brachial plexus injury

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Total/High Spinal Anesthesia

Definition

Extensive spread of local anesthetic within the subarachnoid space that is caused by excessive volume of local anesthetic injected into

the epidural space in a patient with a previous accidental dural puncture, intrathecal injection of local anesthetic following the administration of large volumes of epidural local anesthetic, or injection of a large amount of local anesthetic into the subarachnoid space.

Presentation

- The patient may be conscious but have difficulty speaking or breathing, or the patient may become unconscious.
- The patient will be unable to move her upper extremities.
- Bradycardia is common from blockade of cardiac accelerator fibers (level higher than T1).
- Hypotension caused by extensive sympathetic blockade
- Respiratory distress caused by motor blockade of muscles of respiration.
- Patients with a total spinal anesthetic may have fixed and dilated pupils.

Pathophysiology

Total or high spinal anesthesia is caused by unintentional injection of large amounts of local anesthetic into the subarachnoid space. It may also occur when spinal anesthesia is performed after the administration of large volumes of local anesthetic is administered epidurally, as when an epidural catheter fails to produce adequate anesthesia for cesarean delivery, necessitating a spinal anesthetic. The previously administered epidural local anesthetic compresses the intrathecal space in the lumbar area, causing greater cephalad spread from the intrathecal injection. Another cause of total or high spinal anesthesia is administration of epidural local anesthetic following an accidental dural puncture with an epidural needle. A hyperbaric local anesthetic solution can migrate cephalad if the patient is placed in Trendelenburg position, or a hypobaric solution may rise if the patient is placed into a head-up position.

DIFFERENTIAL DIAGNOSIS

- Myocardial infarction
- Local Anesthetic toxicity
- Vasovagal
- Hemorrhage
- Intracerebral bleed

Immediate Management

- Recognize that the patient has a total or high spinal anesthetic.
- Increase FiO_2 to maintain oxygenation.
- Provide positive pressure ventilation if the patient is in respiratory distress. She may be hand ventilated with a bag/mask or be intubated and ventilated. Although the patient

Immediate Management (*continued*)

may not be responsive, she is awake. A general anesthetic should be started with caution because the patient will also be hypotensive.

- Administer vasopressors (ephedrine or epinephrine) to treat the hypotension. Bradycardia may occur if the level of the block extends beyond T1, which may decrease the effectiveness of phenylephrine.
- Administer additional intravenous fluid.

Diagnostic Studies

No diagnostic studies are indicated for the diagnosis of total/high spinal. If the patient does not respond within the expected time frame following the intrathecal injection, a computed tomography is indicated.

Subsequent Management

- Hypotension resulting from sympathetic blockade may require continuous infusion of a vasopressor such as phenylephrine or epinephrine.
- A low concentration of a potent volatile anesthetic or other sedative should be used to provide amnesia if the patient is intubated.
- If fetal bradycardia occurs, urgent cesarean delivery should be performed.
- Reverse Trendelenburg position is not recommended to prevent further rostral spread, as it will only worsen venous pooling and further decrease venous return to the heart.
- Using mask ventilation until the patient is able to protect the airway is not recommended because it may increase the risk of aspiration and make it difficult to perform other tasks.

Risk Factors

- Short stature
- Spinal anesthesia following failed epidural anesthesia
- Epidural anesthesia following accidental dural puncture

Prevention

- Maintain vigilance while performing neuraxial blocks.
- Reduce the dose of intrathecal local anesthetic in short patients.
- Reduce the dose of local anesthesia in patients with failed epidural anesthesia.

- Always administer a test dose of local anesthetic through the epidural catheter.
- Fractionate the dose of epidural local anesthetic, always soliciting the patient for symptoms of intrathecal injection.

Special Considerations

- Cardiac arrest may occur in patients with high/total spinal anesthesia, with the most common preceding sign being bradycardia.

Further Reading

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Umbilical Cord Prolapse

237

Definition

The umbilical cord descends in advance of the fetal presenting part during labor, protruding into or through the cervix, becoming compressed between the fetal presenting part and the pelvis. This decreases fetal blood flow.

Presentation

Umbilical cord prolapse typically presents with persistent fetal bradycardia or severe variable decelerations in the setting of ruptured membranes. This fetal heart pattern is due to compression of the umbilical cord against the maternal pelvis with decreased oxygen delivery to the fetus.

Pathophysiology

Umbilical cord prolapse occurs when the amniotic membranes are ruptured prior to engagement of the fetus into the pelvis. Two mechanisms have been postulated. The first step is rupture of the amniotic membranes prior to engagement of the fetal presenting part in the maternal pelvis.

- The umbilical cord becomes limp after repeated compression and therefore more easily prolapses.
- Fetal acidosis increases the stiffness of the umbilical cord, which then leads to decreased adaptability and predisposes to cord prolapsed.

DIFFERENTIAL DIAGNOSIS

- Fetal bradycardia
- Placental abruption
- Maternal hypotension
- Oligohydramnios

Immediate Management

- Ask the obstetrician to elevate the presenting part of the fetus, moving it away from the umbilical cord.
- Assess fetal heart rate (ask the obstetrician to palpate the umbilical cord pulsations) to determine whether an emergency cesarean section is necessary.
- Prepare for emergency cesarean delivery. If the patient does not have an epidural catheter in place, consider general anesthesia using rapid sequence induction.

Diagnostic Studies

Consider umbilical cord prolapse in with the setting of fetal bradycardia and ruptured membranes. The diagnosis is confirmed by the obstetrician.

Subsequent Management

If the obstetrician is able to return the umbilical cord back into the uterus and the fetal heart rate improves, it may be possible to perform spinal anesthesia. If an epidural catheter is in place, a local anesthetic with rapid onset of blockade should be used. If no epidural catheter is in place and the fetal heart rate is nonreassuring, general anesthesia may be indicated. The neonate is at high risk for apnea and bradycardia and should be resuscitated by individuals skilled in neonatal resuscitation.

Risk Factors

- Fetal malpresentation
- Preterm delivery
- Low birth weight
- Contracted pelvis
- Multiparity
- Amnioinfusion
- Polyhydramnios
- Twin gestation
- Amniotomy

Prevention

- A careful vaginal examination should be performed prior to rupture of the membranes to insure that the fetus is engaged in the pelvis.
- If the fetus develops bradycardia after membrane rupture, an immediate vaginal examination should be performed rule out prolapse.

Special Considerations

- The incidence of umbilical cord prolapse varies between 0.14% and 0.62% of deliveries and has a high perinatal mortality (approximately 50% due to out of hospital occurrence). If umbilical cord prolapsed occurs in a hospital and a monitored setting, the incidence of perinatal mortality is low (0%–3%). Although spinal anesthesia may be possible, it is technically difficult to position the patient with the obstetrician's hand in the patient's vagina.

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Uterine Rupture

Definition

Uterine rupture is the complete separation of all layers of the uterine wall, resulting in free communication between the uterine and abdominal cavities. Uterine dehiscence is an incomplete disruption of the uterine wall, usually within the serosa overlying the defect in the uterine muscle. A dehiscence is also known as a *uterine window*.

Presentation

- Abdominal pain
 - If an epidural catheter is in place and being used for labor analgesia, the patient may experience breakthrough pain because the concentrations of local anesthetic currently used for the management of labor analgesia are inadequate to cover the pain of uterine rupture.

- Vaginal bleeding
- Loss of fetal station
- Abnormal FHR (usually bradycardia)

Pathophysiology

Uterine rupture is exceptionally rare in women who have not undergone previous uterine surgery, and is thought to be due to uterine anomalies or connective tissue disease. Uterine rupture occurs most commonly when a scar on the uterus begins to separate. After uterine surgery, the risk of rupture depends upon the type of uterine scar. A low transverse scar carries a risk of approximately 1%. The risk associated with a low vertical scar increases to 2%, and further increases to 4%–9% with an inverted T-shaped or classic incision. Uterine rupture may occur following myomectomy. Although rupture generally occurs during labor with uterine contractions, it may also occur before the onset of labor.

DIFFERENTIAL DIAGNOSIS

- Placental abruption
- Placenta previa
- Fetal bradycardia
- Uterine tetany

Diagnostic Studies

There are no diagnostic studies. Suspect uterine rupture in any patient with previous uterine surgery and whose fetus has a Category III fetal heart rate. It also part of the differential in a patient with previous uterine surgery who has breakthrough pain despite an epidural infusion of a low concentration local anesthetic mixture. Ultrasound may be helpful, but is not sensitive for uterine rupture.

Immediate Management

- Prepare for expeditious cesarean delivery.
- Establish large bore intravenous access. There is a high risk of significant hemorrhage.
- Send a maternal blood specimen for type and cross-match.
- Consider the use of a fluid warmer and rapid infusion system.
- Epidural anesthesia may be used, but if the hemorrhage is significant, consider general anesthesia.

Subsequent Management

Uterine rupture is not necessarily an indication for hysterectomy if the uterus can be repaired. If a uterine repair is planned, anticipate

a prolonged procedure and the patient must deliver via elective cesarean delivery. If gravid hysterectomy is performed, close attention to adequate fluid and blood resuscitation is mandatory.

Risk Factors

- Prior uterine surgery (risk increases in direct correlation with number of surgeries)
- Type of scar (classic incision has the highest risk)
- Pregnancy within 2 years of previous cesarean delivery
- Induction of labor in patients with previous cesarean delivery

Prevention

A patient who is considering vaginal delivery after cesarean delivery (TOLAC: trial of labor after cesarean) should not undergo induction of labor with prostaglandins. Prolonged use of oxytocin is associated with uterine rupture. Due to the risk of uterine rupture in patients with a previous cesarean delivery, an anesthesiologist and an obstetrician must be available during any attempted TOLAC in case uterine rupture occurs. This requirement has decreased the number of hospitals that are able to offer TOLAC.

Special Considerations

- Uterine rupture typically occurs in patients who have had previous uterine surgery, although it may rarely occur in a patient who never had previous uterine surgery. Despite the low incidence of uterine rupture during attempted vaginal birth after cesarean delivery, the number of women who attempt vaginal delivery has decreased. A factor in this decrease is the decrease in the number of hospitals where trial of labor after cesarean section is offered. The ability to have an immediately available obstetrician and anesthesiologist limits the ability to offer TOLAC.

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Chapter 9

Pediatric Emergencies

Anna Clebone and Bradley Besson

Anaphylaxis	244
Asthma and Bronchospasm (Lower Airway Obstruction)	247
Burns	250
Epiglottitis (Supraglottitis): Nonperioperative Onset of Stridor	256
Inhaled Foreign Body	260
Complete Airway Obstruction (No Air Movement)	260
Near-Drowning	262
Neonatal Resuscitation	265
Pediatric Advanced Life Support: Outside the Operating Room	269
Stridor, Postextubation	274
Trauma	276

Critical event checklists, developed by the Society of Pediatric Anesthesia, Quality and Safety Committee, can be found at:

http://www.pedsanesthesia.org/newnews/Critical_Event_Checklists.pdf?201310291500

and as an iPhone app:

<https://itunes.apple.com/us/app/pediatric-critical-events/id709721914?ls=1&mt=8>

Anaphylaxis

Definition

A rapid, possibly life-threatening allergic reaction caused by the sudden release of inflammatory mediators after exposure to an allergen, most commonly neuromuscular blockers, antibiotics, or latex.

Presentation

In an anesthetized patient:

- Erythematous rash (may be absent in quickly progressive anaphylaxis) Wheezing, difficult ventilation, and hypoxia caused by bronchospasm
- Face, lip, tongue, and laryngeal edema. Stridor may be present in a patient who is not intubated.
- Hypotension (may be the only sign of anaphylaxis in the anesthetized patient)
- Tachycardia or bradycardia (the latter occurs in 10% of patients, often associated with severe hypovolemia)
- Cardiac arrest

Additional signs and symptoms in an awake patient:

- Shortness of breath, tachypnea, use of accessory respiratory muscles
- Itching
- Nausea, vomiting, and abdominal pain
- Dizziness or altered mental status
- Hoarseness or stridor caused by laryngeal edema

Pathophysiology

Anaphylaxis is an immediate, life-threatening hypersensitivity reaction to a specific antigen, affecting multiple organ systems, and caused by IgE activation of mast cells and basophils and release of inflammatory mediators. Increased vascular permeability can cause a 35% decrease in circulating blood volume in 10 minutes, causing hypovolemia and shock. Arterial vasodilation produces decreased systemic vascular resistance and tissue hypoperfusion.

Immediate Management

- **Identify and discontinue antigens** (e.g., antibiotic, colloid, drug infusion). Remove latex surgical equipment or urinary catheter. Wash area if latex or chlorhexidine allergy is suspected.
- Administer supplemental oxygen or increase FiO_2 to 100%.
- Intubate the trachea if airway obstruction is imminent or if the patient is hypoxemic. **Progressive laryngeal edema may make late endotracheal intubation or cricothyroidotomy impossible.**
- Epinephrine, 0.1–3 MICROgrams/kg intravenously (IV) (a fraction of the cardiac arrest dose!) depending on the severity of reaction, repeated if needed. Titrate carefully to avoid adverse hemodynamic consequences.
- Establish large-bore IV access.
- Begin aggressive resuscitation with IV fluids to support intravascular volume (10–20 mL/kg, repeat until blood pressure stabilizes).
- Decrease or discontinue anesthetic agents as necessary to maintain blood pressure.
- Treat bronchospasm with aerosolized albuterol. If intubated: Administer 4–10 puffs from a metered dose inhaler through an endotracheal tube. If unintubated: Administer 0.15 mg/kg (minimum of 2.5 mg, max 10 mg) of 0.083% albuterol diluted in 3 cc normal saline through a nebulizer.
- Potent volatile anesthetic agents (if tolerated hemodynamically) and IV ketamine may reduce bronchospasm.
- Rarely, catecholamine resistant anaphylactic shock can occur, necessitating treatment with vasopressin, or, infrequently, methylene blue.

DIFFERENTIAL DIAGNOSIS

- Rash: Mild or localized allergic reaction
- Bronchospasm: Bronchial hyper-reactivity from recent upper respiratory infection, acute asthma exacerbation, aspiration (of a foreign body or gastric contents)
- Hypotension: Blood transfusion reaction, red man syndrome from rapid administration of vancomycin, mastocytosis (In the latter, patients often have had previous hemodynamic events, will have negative skin tests, and tryptase is increased at baseline.)

Diagnostic Studies

- Anaphylaxis is diagnosed based on clinical manifestations; at least two organ systems must be involved to make the diagnosis.
- Analysis of a blood sample for histamine (send quickly; half-life is 15–20 minutes) and mast cell tryptase (half-life is 2 hours) may confirm the diagnosis.

Subsequent Management

- After a severe reaction, begin a continuous infusion of epinephrine 0.01–0.2 MICROgrams/kg/min.
- Administer diphenhydramine (H_2 blocker) IV 1 mg/kg, maximum 50 mg and famotidine 0.25 mg/kg IV OR ranitidine (H_1 blocker) 1 mg/kg IV.
- Administer dexamethasone 0.2–1 mg/kg IV, or methylprednisolone 2 mg/kg IV, maximum 100 mg to decrease inflammation (effect occurs in 4–6 hours).
- If a severe reaction occurs, discuss canceling or limiting the surgical procedure.
- Refer to an allergist for skin testing in 4–6 weeks.

Risk Factors

Latex allergy is more common in children with spina bifida or other genitourinary abnormalities due to multiple exposures during surgery and catheterization.

Prevention

Obtain a detailed history of previous allergic reactions, atopy, and asthma. Question patients specifically about latex allergy, including allergy to foods with cross-reactivity (e.g., banana, kiwi, papaya, avocado) and avoid latex in those patients.

Prophylactic medications to prevent anaphylaxis are not recommended because they may mask a true reaction and delay immediate diagnosis and treatment.

Special Considerations

- Epinephrine is a drug with a narrow therapeutic index, and the dose to treat anaphylaxis is smaller than for cardiac arrest. Epinephrine overdose may cause severe hypertension, potentially causing cerebral or myocardial injury, pulmonary edema, and ventricular dysrhythmias.
- Avoid atropine administration for bradycardia, especially if the patient is hypovolemic. Treat bradycardia in the setting of

anaphylaxis with fluid resuscitation followed by epinephrine. Bradycardia may be protective, allowing the ventricles to fill in the setting of massive hypovolemia.

- Between 5% and 20% of patients will have a recurrence of anaphylaxis 8–12 hours after initial presentation.
- Respiratory abnormalities are the most common symptom of anaphylaxis in children (as opposed to adults, in whom cardiovascular instability is more common).
- Breath sounds may be absent in a patient with severe bronchospasm because of decreased air exchange.
- A rapid onset of anaphylaxis is associated with a more severe or possibly fatal reaction.

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247

Asthma and Bronchospasm (Lower Airway Obstruction)

Definition

Increased resistance of the airways caused by hyperreactivity, inflammation, edema, and mucous plugging.

Presentation

In an anesthetized patient:

- Wheezing
- Diminished or absent breath sounds (indicates severe bronchospasm and minimal airflow)
- Increased peak airway pressure
- Hypoxemia

Additional signs and symptoms in an awake patient:

- Dyspnea, increased work of breathing, use of accessory muscles, retractions, nasal flaring, “tripod” position
- Tachypnea with a prolonged expiratory phase
- Diaphoresis
- Cough with sputum production
- Chest pain or tightness and anxiety

- Late bronchospasm: Obtundation, respiratory failure, and cardiac arrest

Pathophysiology

Airway obstruction leads prolonged expiratory time (increased I:E ratio) in the spontaneously breathing patient. If the I:E ratio is not manually increased in the mechanically ventilated patient, air will become trapped in the alveoli, causing hyperinflation.

Immediate Management

- Increase FiO_2 to 100%.
- Increase the inspired concentration of potent volatile anesthetic and/or administer propofol to increase the depth of anesthesia. (Note: Desflurane is a bronchial irritant. Switch to sevoflurane if possible.)
- Check the position of the endotracheal tube; consider the possibility of mainstem intubation, kinking, or circuit disconnection.
- Suction the endotracheal tube.
- Administer albuterol (β_2 -agonist). If intubated: 4–10 puffs from a metered dose inhaler through the endotracheal tube. If unintubated, 0.15 mg/kg (minimum of 2.5 mg, max 10 mg) of 0.083% albuterol diluted in 3 cc normal saline through a nebulizer.
- Administer corticosteroids (dexamethasone 0.1–1 mg/kg IV; reduces inflammatory sequelae; effects in 4–6 hours).

If the preceding are ineffective, attempt the following:

- Administer ketamine 1–2 mg/kg IV (bronchodilator) with atropine or glycopyrrolate (0.02 mg IV increments titrated to heart rate) to decrease secretions. CAUTION if unintubated. Ketamine will cause sedation.
- Administer epinephrine 1 MICROgram/kg IV.
- Consider extracorporeal membrane oxygenation (ECMO) in a patient with unremitting, life-threatening bronchospasm. Note: Alert cardiac surgical and perfusion teams early if the need for ECMO is anticipated. Caution: ECMO is resource intensive and carries many risks and therefore should be used as a last resort.

DIFFERENTIAL DIAGNOSIS

- Atelectasis
- Anaphylaxis (is accompanied by rash and hypotension)
- Pulmonary edema caused by fluid overload (especially in an infant)

- Foreign body aspiration (e.g., a tooth that is dislodged during laryngoscopy and intubation)
- Pulmonary aspiration of gastric contents
- Tracheomalacia or bronchomalacia, especially in a former premature infant

Diagnostic Studies

- Chest radiograph may show hyperinflation of the lungs caused by air trapping
- Arterial blood gas in decompensating patients. Multiply FiO_2 by 5 to estimate expected “normal” PaO_2 .
- A spontaneously breathing patient who is hyperventilating will first develop a respiratory alkalosis. Later on, increasing or “normal” PaCO_2 may indicate imminent respiratory failure.

Subsequent Management

- Administer oral steroids for several days to a week after surgery in moderate to severe cases.
- If respiratory status does not normalize, delay extubation and/or admit the patient to the pediatric intensive care unit (PICU).

Risk Factors

- Pre-existing upper respiratory infection (URI) or reactive airway disease/asthma increase the risk of bronchospasm. The airway will be hyper-reactive for 6 weeks after a URI.
- Bronchospasm can be caused by endotracheal intubation or other airway instrumentation.
- Atopy, including pre-existing asthma, environmental allergies, and eczema.

Prevention

- Postpone elective surgery if a patient is wheezing preoperatively. For emergency surgery, administer inhaled albuterol in the preoperative area and advise the parents that extra oxygen, a prolonged postoperative stay, or prolonged intubation and PICU admission may be required.
- Continue home medications, including inhaled corticosteroids (fluticasone) and leukotriene inhibitors (monteleukast), in patients with known reactive airway disease or asthma. If a patient is at high risk for intraoperative bronchospasm, several days of oral steroids (e.g., prednisone 1 mg/kg/day) should be administered before surgery.

Special Considerations

- Airway instrumentation and endotracheal intubation can precipitate bronchospasm by irritating the airways. It may be possible to avoid bronchospasm by using mask ventilation or a laryngeal mask airway for the duration of the anesthetic. If the trachea will be intubated, removing the tube during stage 3 of anesthesia (i.e., a “deep” extubation) may decrease bronchospasm, but may increase the risk of laryngospasm during emergence.

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Burns

Definition

Thermal injury that compromises the skin, airway, and other organs, predisposing to infection and impairing the body's ability to regulate temperature and maintain fluid and electrolyte balance. Extensive local tissue damage can lead to hypermetabolism and multiorgan failure. First-degree burns involve only the epidermis, second-degree burns cross into the dermis, third-degree burns involve the subcutaneous tissue, and fourth-degree burns involve the bone or muscle.

Presentation

- First degree: painful, red skin. Second degree, Superficial: erythema, pain, blisters. Second degree, Deep: white, leathery skin, less pain (indicates nerve damage). Third degree: white, leathery, painless. Fourth degree: may be fatal. Bone and muscle are involved. Tissue injury may be worse than surface appearance in electrical or chemical burns.
- If severe, systemic manifestations:
- Airway: Progressive edema and airway obstruction over the first 24 hours are caused by smoke inhalation injury and resultant

inflammation. This is indicated by burnt nose hairs, facial burns, ashes around the mouth or soot in the sputum.

- **Pulmonary:** Dyspnea, bronchospasm, stridor, hypoxemia. Toxin damage to the tracheobronchial tree is symptomatic 12–36 hours post injury. **Carbon monoxide (CO) poisoning** may be present despite normal SaO_2 . Hydrogen cyanide (HCN) poisoning can cause severe metabolic acidosis. Pulmonary injury, CO toxicity, or HCN poisoning can occur without external injuries.
- **Cardiac:** Decreased cardiac output, hemoconcentration, and edema caused by fluid shifts into the burned tissue. Myocardial depression. Hypoperfusion and hypotension leading to metabolic acidosis and end-organ damage.
- **Neurologic:** Hypoxic encephalopathy (hallucinations, seizures, coma). Chemical inhalation or CO poisoning can cause altered mental status.
- **Renal:** Injury caused by hypoperfusion, myoglobinuria, or hemoglobinuria.

Pathophysiology

- The airway can be injured by thermal injury from steam, smoke particles, chemicals, and gases that are toxic. Necrotic respiratory epithelium will begin shedding approximately 3 days after injury and may cause airway obstruction.
- Hypothermia is caused by fluid evaporation from injured tissue. Hypothermia may develop quickly in children because the body surface area is high relative to height. Hypermetabolism occurs in proportion to the severity of injury, and involves the hypothalamic mediated release of glucagon, cortisol, and catecholamines. Catecholamine and vasoactive mediator release cause a capillary leak around the burned tissue during the first day after injury.

DIFFERENTIAL DIAGNOSIS

- Acute respiratory distress syndrome (may indicate progression of inhalation injury).
- Stevens-Johnson syndrome: A severe, red, blistering rash and epidermal necrosis that occurs as an immune-mediated reaction to systemic infection or certain medications.

Immediate Management

Treat Life-Threatening Systemic Conditions

- Intubate the patient immediately with a microcuffed endotracheal tube if the patient has signs of an inhalation injury (see Special Considerations) or facial burns. Advanced

Immediate Management (continued)

airway equipment (fiberoptic, glidescope) must be available. Caution: Succinylcholine may be used only within 24 hours of the burn. Mask ventilation may be difficult if the face is burned.

- Administer 100% oxygen. If possible, humidify inspired gases to minimize irritation.
- Establish large-bore IV access. Begin aggressive fluid resuscitation according to the Parkland formula. Titrate urine output to 0.5–1 mL/kg/h.
- If necessary, support hemodynamics with inotropic agents (can have depressed cardiac function).
- Prevent hypothermia by increasing the room temperature. Cover the patient with a warming blanket. Warm all fluids.
- If HCN poisoning is suspected, immediately administer hydroxocobalamin IV 70 mg/kg (max 5 g).
- Remove all affected clothing and begin a primary trauma survey (see Major Trauma, page 276).
- Apply cold, running water (12–18° C) to the burned tissue for at least 20 minutes for analgesia and to reduce burn depth. Caution: Avoid hypothermia. This should be done as soon as possible, but may be beneficial for up to 3 hours after injury.
- Cover the burn with clear plastic wrap or sterile plastic bags to reduce fluid and heat loss.
- Administer analgesia as tolerated. Intranasal fentanyl can be given if intravenous (IV) access is not yet established, but monitor carefully for apnea.

Diagnostic Studies

- Treatment decisions are partially based on the percentage of total body surface area (TBSA) injured (blistering or worse). Children have relatively smaller limbs and bigger heads than teenagers or adults, which alters this calculation (see Figure 9.1, Rule of 9s).
- Complete blood count, electrolytes creatine kinase, urine myoglobin (rhabdomyolysis), co-oximetry (CO poisoning).
- Serial arterial blood gas measurements guide ventilation and oxygenation (PaCO_2 , PaO_2) and treatment of metabolic acidosis.
- Chest radiograph demonstrates lung opacities in severe inhalation injury. (Lungs will appear normal in less severe inhalation injury.)

Subsequent Management**Fluid Resuscitation**

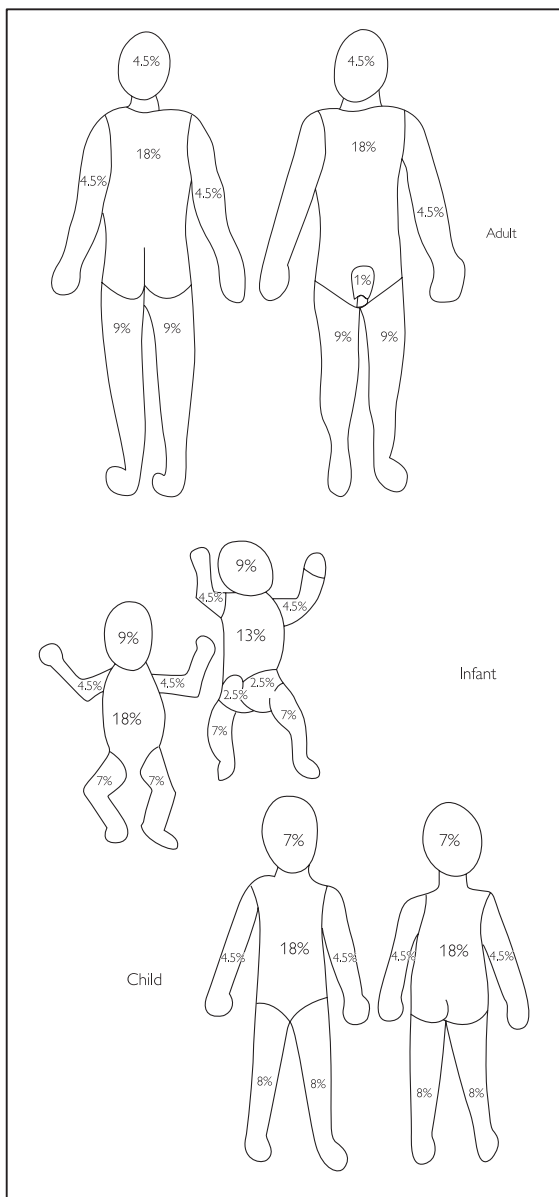


Figure 9.1 Rule of Nines: Body Surface Area in Adults versus Children

- Estimate crystalloid fluid requirements with the Parkland formula: $4 \text{ mL/kg} \times \text{percent burn} \times \text{wt (kg)} = \text{first 24-hour additional requirements (beyond maintenance)}$, with half given in the first 8 hours. Can be up to one-third higher if a concomitant inhalation injury exists. Titrate IV fluids to vital signs, urine output ($0.5\text{--}1 \text{ mL/kg/h}$), pulse oximeter and arterial line respiratory variations, central venous pressure.
- Add glucose to IV fluids if needed, especially in younger patients.
- If necessary, administer colloids (albumin 5%) to keep serum albumin $>2.0 \text{ g/dL}$.
- Add potassium to IV fluids as necessary to compensate for loss in the urine or from burned tissues.
- Administer blood products as indicated for anemia or coagulopathy.
- Consider inserting a central venous catheter in patients with burns of $>30\%$ of the body's surface area.

Breathing

- Check endotracheal tube cuff pressures frequently. Maximum airway edema typically occurs 24 hours after injury, and then decreases. It may be necessary to secure the endotracheal tube with cloth ties because tape will not stick to injured tissue.
- Circumferential, deep burns to the abdomen and chest may impair ventilation by limiting chest wall excursion, and may require urgent surgical escharotomy.

Toxicity

- In patients with severe HCN toxicity, consider administering sodium thiosulfate 1.65 cc/kg IV to a maximum of 12.5 g , in addition to hydroxocobalamin (may be synergistic)

Neurologic

- Multimodal pain management: Consider fentanyl and hydromorphone (less histamine release than morphine), ketamine, midazolam, and dexmedetomidine. Patients may need continuous narcotics and sedation. Tolerance develops quickly; doses will need to be increased over time.

Risk Factors

- Half of pediatric burns occur in children <5 years old.
- Twenty percent of pediatric burns are due to child abuse. Glove and stocking burns, especially without splash marks and if full thickness, or on the soles of both feet or on the backside should raise the suspicion of abuse.

Special Considerations

- Patients are at increased risk of aspiration due to delayed gastric emptying or recent oral intake. They are also at risk for difficult ventilation and intubation due to inflammation and swelling. An inhalation induction, maintaining spontaneous ventilation until the airway is secured, may be safer if the airway is compromised. Note: Equipment necessary to establish a surgical airway should be immediately available. Under-resuscitation or delayed fluid resuscitation will lead to tissue hypoperfusion, acidosis, and increased mortality. Over-resuscitation with intravenous fluids can lead to increased tissue swelling, compartment syndrome, and even pulmonary edema.
- Intravenous access may be difficult. It may be necessary to insert a catheter through burned tissue to avoid delays in resuscitation. Consider an intraosseous catheter or venous cutdown if adequate access cannot be obtained.
- Inhalation injury can occur even in the absence of skin burns and should be suspected if soot is present in or around the airway. Emergency airway equipment should be immediately available, and the team should be ready to manage a possible difficult airway. Secure the airway immediately if respiratory distress, severe burns to the face or neck, or swelling or blistering inside the mouth are present. If ulcerating burns are present in the airway, an early tracheostomy is indicated to avoid further airway trauma. Even if immediate intubation is not indicated, maintain a high level of suspicion for airway injury. If steam inhalation is suspected, perform a fiberoptic bronchoscopy to assess the degree of thermal injury of the tracheobronchial tree. Caution: Laryngoscopy or bronchoscopy can precipitate laryngospasm, bleeding, or swelling and subsequent airway obstruction. Younger children will not tolerate these procedures and may require general anesthesia.
- Consider carbon monoxide or hydrogen cyanide poisoning in patients with smoke inhalation (may occur in up to 35% of patients). SaO_2 will not be decreased in CO poisoning because a standard pulse oximeter cannot differentiate between oxyhemoglobin and carboxyhemoglobin. Carbon monoxide poisoning should be suspected if there was a prolonged exposure to smoke or the patient has mental status changes, chest pain (from hypoxia), headache, or nausea. Send blood for co-oximetry on blood gas or use a noninvasive pulse co-oximeter to diagnose CO inhalation. A child with chronic exposure to tobacco smoke will have an already elevated CO level of approximately 1.5%. Consider hyperbaric oxygen if the CO level is $>25\%$, there are mental status changes, signs

of end-organ injury, or myocardial ischemia, or if pH is <7.1 . HCN is present in smoke when materials containing both carbon and nitrogen are burned. HCN poisoning presents with stomach pain, mental status changes, and an initially elevated respiratory rate, heart rate, and blood pressure followed by cardiopulmonary collapse, rhabdomyolysis, and multi-system organ failure. Laboratory studies will reveal an anion gap metabolic acidosis, elevated lactate (often >10 mmol/L), a decreased venous to arterial PO_2 gradient, and a blood cyanide level. Amyl nitrite and sodium nitrite can cause methemoglobinemia and should not be used if concomitant CO poisoning is suspected. Patients may also require intubation and mechanical ventilation and hemodynamic support with vasopressors. Caution: Co-oximetry may be inaccurate after the patient has received hydroxycobalamin.

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Epiglottitis (Supraglottitis): Nonoperative Onset of Stridor

Definition

Epiglottitis is a relatively rare, but immediately life-threatening cause of stridor that can lead to rapidly progressive airway obstruction (Table 9.1).

Presentation

Airway

- Classic appearance of “4Ds”: drooling, dyspnea, dysphonia (muffled voice), and dysphagia. The patient may complain of a severe sore throat, and will have inspiratory stridor.

Breathing

- Normal respiratory rate

Systemic

- High fever often $>39^\circ$ C, toxic appearance

Table 9.1 Nonoperative Onset of Stridor in Children

	Croup (laryngotracheo- bronchitis)	Epiglottitis	Inhaled Foreign Body
Incidence	Common (>80% of stridor)	Rare	Varies
Fever	High	High	None
Acuity	Often benign	Can be life-threatening	Can be life-threatening
Onset	After 12–24 hours of rhinitis and “cold” symptoms	Sudden without prodrome	Sudden
Cough	Common (due to involvement of the trachea and bronchi) and high-pitched (due to turbulent air flow through the narrowed subglottis)	Rare	Common
Drooling	Absent	Typical	No
Dysphonia	Hoarse	Muffled	
Dysphagia	Sometimes	Severe	
Dyspnea	Present	Present	Present
Stridor	Loud inspiratory stridor (due to inflammation and edema at the extrathoracic narrowed cricoid ring) Fast respirations (due to the involvement of the trachea and bronchi)	Soft inspiratory stridor (epiglottis is extrathoracic , but airflow is decreased due to inflammation)	Expiratory (or biphasic) stridor if foreign body is in the bronchus, due to intrathoracic obstruction
Age	<3	2–8	Varies
Radiography (only in a stable patient)	Tracheal narrowing “steeple sign”	Epiglottis swollen “thumbprint sign”	If object is radio-opaque, often seen in bronchi or trachea
Intervention	Humidified oxygen Racemic epinephrine Intravenous steroids	To operating room, intubate, ICU, antibiotics	To operating room, inhalation induction, remove with rigid bronchoscopy by ENT

- Patient may be found sitting up and leaning forward with the chin protruding in a sniffing position.

Pathophysiology

Epiglottitis is an inflammatory edema of the supraglottic structures, including the arytenoids, aryepiglottic folds, uvula, and epiglottis, commonly caused by *Haemophilus influenzae* type B. Other bacteria (e.g., Group A β -hemolytic streptococcus, staphylococcus aureus), viruses (e.g., herpes simplex, varicella-zoster), and mechanical trauma or thermal injury can also cause epiglottitis.

Immediate Management

- If complete airway obstruction is imminent, call personnel skilled in advanced airway management and an otorhinolaryngologist to the bedside.
- Administer O₂ via facemask or “blow-by” as tolerated. Avoid airway examination or manipulation (may precipitate laryngospasm).
- Transport the patient to the operating room. An anesthesiologist and otolaryngologist should remain with the patient at all times. Advanced airway management equipment, including several endotracheal tubes of expected size for age and smaller, and stylets, should be immediately available during transport.
- If possible, allow a parent to accompany the patient to the operating room to minimize agitation and the risk of airway compromise. Allow the patient to remain in the sitting position. Avoid painful procedures (IV placement) until anesthetized in the operating room.
- Ensure that all equipment necessary for a possible rigid bronchoscopy or surgical airway is immediately available.
- Induce general anesthesia via inhalation with sevoflurane, even in a patient with a full stomach. Keep the child in the sitting position on the parent’s lap during induction. Maintain spontaneous ventilation with a deep plane of anesthesia (spontaneous ventilation may facilitate location of the vocal cords in a patient with an edematous airway). Caution: **Do not** use intravenous agents or muscle relaxants until the airway is secured.
- Establish intravenous access after patient is adequately anesthetized.
- Carefully perform an endotracheal intubation, lifting the tongue gently without touching or traumatizing the epiglottis.

DIFFERENTIAL DIAGNOSIS

- Croup (more common, 80% of children with stridor)
- Foreign body in the airway or esophagus (history of choking while eating; sudden, persistent cough)
- Retropharyngeal abscess

Diagnostic Studies

- Primarily a clinical diagnosis. **DO NOT DELAY TREATMENT.**
- Lateral neck radiograph can show an enlarged epiglottis appearing as a “thumbprint,” obscuring the vallecula (Note: This is not required for diagnosis and should only be attempted in stable patients.) Ultrasonography can show an “alphabet P sign.”
- After securing the airway, blood cultures and throat cultures can assist in identifying the causative organism and antibiotic sensitivities.

Subsequent Management

- Intubation may be required for 24–36 hours until airway inflammation is resolved. Signs of decreased swelling include an air leak around the cuff of the endotracheal tube after deflation.
- Humidify inspired gases.
- Transfer to the intensive care unit.

Risk Factors

- Winter months
- Immunosuppression

Prevention

Universal vaccination to decrease H. flu infection.

Special Considerations

- Late complications of epiglottitis can include subglottic granulomas or stenosis, as well as tracheomalacia.

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Inhaled Foreign Body

Definition

The presence of a foreign body in the hypopharynx, larynx, trachea, or lungs, or posterior compression of the trachea caused by an object obstructing the esophagus.

Presentation

- A witnessed choking episode may be reported.
- Stridor, shortness of breath, coughing, wheezing
- Late presentation could be a chronic nonasthmatic wheeze or persistent pneumonia
- If severe obstruction of trachea or both bronchi, child may be cyanotic.

Immediate Management

Partial Airway Obstruction

- Monitor respiratory status closely.
- Transport the patient to the operating room. The patient should be transported by personnel skilled in airway management and emergency airway equipment must be immediately available.
- Perform an inhalation induction of general anesthesia. Keep the patient spontaneously breathing.
- To facilitate rigid bronchoscopy, begin a propofol infusion (200–300 MICROgrams/kg/min or as tolerated) to maintain spontaneous respirations on 100% O₂ and provide a deep plane of anesthetic (so the patient does not move or cough, which could cause airway injury).
- If the airway is injured or edematous, intubate the trachea after the foreign body has been removed.

Complete Airway Obstruction (No Air Movement)

- If the patient is awake, perform the Heimlich maneuver.
- Request an emergency otolaryngology consult to perform direct laryngoscopy and rigid bronchoscopy (to attempt to stent the airway open and either remove the object or push the object distally so that air movement can occur).

DIFFERENTIAL DIAGNOSIS

- Acute asthma exacerbation (wheezing, respiratory distress, prolonged expiratory phase).
- Croup (gradual onset, barking cough, hoarseness, rapid respiratory rate).
- Foreign body in the esophagus (drooling, dysphasia, dyspnea).
- Epiglottitis (acute onset, toxic appearance, sore throat, stridor).
- Anaphylaxis (sudden onset, exposure to allergen, urticaria, possible hypotension).
- Retropharyngeal abscess (airway obstruction, stridor).

Diagnostic Studies

- Radiography may be nondiagnostic and should be attempted only in a stable patient. Eighty-nine percent of foreign bodies may not be visualized on radiography, and sequelae (e.g., hyperinflation or atelectasis) may not be seen in 17% of patients.

Subsequent Management

- Unless otherwise contraindicated, administer a steroid (e.g., dexamethasone 0.5–1 mg/kg) to decrease late inflammatory edema.
- Treat bronchial hyper-reactivity and resultant wheezing (from the foreign body and from instrumentation) with inhaled albuterol (4–10 puffs from a metered dose inhaler through an endotracheal tube, or 0.15 mg/kg [minimum of 2.5 mg] of 0.083% diluted in 3 cc normal saline through a nebulizer for an intubated patient).
- Upper airway irritation causing stridor can be treated with nebulized racemic epinephrine 0.5 mL of 2.25% solution diluted in 2 mL normal saline (0.3 mL for infants).

Risk Factor

- Age <4 years

Prevention

Check the mouth carefully for loose teeth prior to laryngoscopy, especially in young or developmentally delayed patients.

Special Considerations

- Removal of the foreign body in the operating room with full anesthesia and surgical equipment including a rigid bronchoscope and tracheostomy kit is the safest option.

Agitation, multiple attempts at removal, positive pressure ventilation, and endotracheal intubation may cause the object to move, rapidly converting partial airway obstruction into complete airway obstruction.

- Spontaneous ventilation should be maintained because intravenous anesthetic agents or neuromuscular blocking agents may cause relaxation of airway muscles and lead to complete obstruction.
- Complications that may occur during removal of the foreign body include aspiration, bronchospasm, severe edema of the larynx, laceration of the trachea or bronchi, pneumothorax, pneumomediastinum, or hypoxia.

Further Reading

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Near-Drowning

Definition

Interruption of breathing due to immersion in liquid. Pulmonary, cardiac, neurologic, and global end-organ injury may occur due to hypoxia and hypothermia (see chapter 5). Presentation may be asymptomatic or catastrophic, depending upon on how quickly the patient was rescued.

Presentation

Pulmonary

- Hypoxemia
- Apnea when body temperature falls to $<28^{\circ}\text{C}$.

Cardiac

- Dysrhythmias or cardiac dysfunction (a result of hypoxemia, hypothermia, or acidosis). Osborn (J) waves on electrocardiogram may be present when core temperature is $<32^{\circ}\text{C}$. Cardiac arrest caused by myocardial ischemia.
- Hypovolemia due to brisk diuresis (hypothermia decreases antidiuretic hormone production early in submersion).

Neurologic

- Altered mental status (core temperature $<32^{\circ}\text{C}$), coma (core temperature $<30^{\circ}\text{C}$).

Pathophysiology

Water aspiration causes loss of surfactant and atelectasis. Pulmonary edema and damage to alveoli may increase intrapulmonary shunt to 75%. Patients commonly develop acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) after a near-drowning episode. Hypoxic encephalopathy may also occur.

DIFFERENTIAL DIAGNOSIS

- Spinal cord injury (e.g., after diving in shallow water)
- Head injury after a slip and fall accident near water
- Toxic ingestion (e.g., alcohol poisoning, especially in adolescents)
- Seizure

Immediate Management

- Immobilize the cervical spine if head or neck trauma is suspected.
- Clear debris and fluid from the airway.
- Assess airway, breathing, circulation, perform pediatric advanced life support (PALS) if indicated (see page 269).
- Initiate mask ventilation if the patient is apneic; begin chest compressions if pulseless.
- Measure rectal temperature. Do not discontinue PALS until the patient is rewarmed to at least 32° C.
- Evaluate for associated traumatic injuries.

Diagnostic Studies

- Monitor for late end-organ consequences of hypoxia: GI bleeding, coagulopathies, myoglobinuria, hemoglobinuria, and renal failure.
- Arterial blood gas, electrolytes, and toxicology screen (including ethanol)
- Chest radiograph

Subsequent Management

Systemic

- Rewarm the patient: Use forced air warming, radiant warming (warming lights), warm, humidified O₂, and a fluid warmer. Caution: Monitor blood pressure carefully because vasodilation may cause hypotension. If temperature is very low (<32° C), consider warming with cardiopulmonary bypass or hemodialysis.

Cardiac

- In patients who are in cardiac arrest: Continue advanced cardiac life support (ACLS) at least until the core temperature has reached 37° C.

Respiratory

- Treat bronchospasm with albuterol (4–10 puffs from a metered dose inhaler through an endotracheal tube, or 0.15 mg/kg (minimum of 2.5 mg) of 0.083% diluted in 3 cc normal saline through a nebulizer in an intubated patient. Patients with severe hypoxia may require CPAP (continuous positive airway pressure). If the patient is not intubated, consider using a BiPAP mask. Consider PEEP (positive end-expiratory pressure) in an intubated patient to decrease ventilation-perfusion mismatch.

Special Considerations

- Acute respiratory distress syndrome and pulmonary edema can occur up to 24 hours after a near-drowning episode, even in children who first present without apparent injury. Maintain a low threshold for overnight admission.
- Hypoxia for >5–10 minutes is associated with a worse outcome, as are laboratory values showing a very low pH, high lactate, and high blood sugar.

Risk Factors

- Healthy children ages 1–4 who cannot swim and who are not closely supervised.
- Children with a seizure disorder or developmental delay.
- Hypothermia from swimming in cold water in a child with long QT syndrome (body temperature <35° C increases the QT interval).

Further Reading

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Neonatal Resuscitation

Definition

Factors such as prematurity (birth at <37 weeks postconceptual age), congenital abnormalities, peripartum fetal asphyxia, or the failure to warm and dry the newborn can impair the transition from fetal to neonatal circulation and may require advanced resuscitation measures.

Presentation

- At birth, assess for the following: Born full term? Breathing or crying? Good muscle tone? If the answer to any of these questions is “no,” proceed immediately to the Immediate Management section.

If in distress, may also see:

- Blue, floppy, minimal movement, ineffective or absent respirations
- Bradycardia (heart rate <100 beats per minute [bpm]). In children and neonates, hypoxia is the most common cause of bradycardia.
- See Table 9.2 for APGAR score.

DIFFERENTIAL DIAGNOSIS

Rare conditions that may cause neonatal cardiovascular collapse:

- Hypovolemia from hemorrhage before birth
- Diaphragmatic hernia (difficult to ventilate, scaphoid abdomen, bowel sounds present on auscultation of the chest)
- Hydrops fetalis (leading to hemolytic anemia)
- Congenital complete heart block (suspect this when heart rate does not increase to >60 bpm)

Table 9.2 Apgar Score

Clinical Feature	0	1	2
Color	Pale/blue	Acrocyanosis (Pink body, blue extremities)	Pink
Heart rate (beats/minute)	Absent	<100	>100
Response to stimulation	None	Grimace	Pulls away, crying
Muscle tone	Limp, flaccid	Some flexion of extremities	Active movement
Respiratory rate	Absent	Poor effort, slow, irregular	Good, crying

- Pneumothorax (may occur during resuscitation, decreased breath sounds)
- Residual drug effects or withdrawal (i.e., opioids administered to the mother prior to delivery or maternal addiction to drugs or alcohol)

Immediate Management

First 30 seconds:

- Warm and dry the infant.
- Suction the airway if needed.
- Stimulate (rub the back, tap the feet).
- Cover a preterm baby's torso and limbs with plastic wrap to maintain heat.

Second 30 seconds:

- Evaluate heart rate (auscultate or feel for an umbilical pulse), respirations, and color
- If heart rate is <100 or infant is gasping or apneic, begin positive pressure ventilation (rate of 40–60 breaths/minute) and monitor SaO_2 . Begin with room air and increase FiO_2 as needed. Caution: Avoid high peak pressures.
- If breathing is labored or the neonate remains cyanotic, suction the airway, monitor SaO_2 , and consider CPAP.

Third 30 seconds (after 30 seconds of adequate ventilation):

- If the heart rate is <60 , begin chest compressions. Place two thumbs on the lower one-third of the sternum. The depth of compressions should be one-third of the chest while maintaining a rate of 120/min. Give three chest compressions for each breath. The mnemonic for this is: "one-and-two-and-three-and-breathe-and"

Persistent heart rate <60 bpm:

- Administer epinephrine 0.01–0.03 mg/kg IV (preferred) or 0.1 mg/kg via the endotracheal tube. Repeat every 3–5 minutes.
- Intubate the trachea if the chest does not rise with positive pressure ventilation. A supraglottic airway (laryngeal mask airway) can be used as a rescue device.
- Confirm endotracheal tube position above the carina by looking for bilateral chest rise. Mainstem intubation is common in newborns, and is not reliably detected by auscultation. Avoid peak airway pressures >20 – 25 cm H_2O .
- Consider the possibility of complications such as pneumothorax (treat with needle thoracostomy) or hypovolemia (administer fluid or blood with 10 cc/kg boluses).

Immediate Management (continued)

- Assess respirations, heart rate, and color every 30 seconds. Continue coordinated chest compressions and ventilations until spontaneous heart rate is >60 bpm.
- Consider administering surfactant through the endotracheal tube (beractant 4 mL/kg intratracheal) for severe respiratory distress in preterm infants (especially if <28 weeks gestation). Divide into four aliquots and administer in different positions to ensure adequate distribution.
- Infants with meconium in their amniotic fluid require intubation and suctioning of the endotracheal tube only if they do not successfully transition to postpartum physiology (if the infant has a heart rate <100 bpm with poor muscle tone and respiratory effort).

Diagnostic Studies

Do not stop ventilation or chest compressions to obtain laboratory or diagnostic studies. Treat suspected pneumothorax with a needle thoracostomy based on clinical suspicion. If possible, resuscitation may be guided by arterial blood gas analysis, complete blood count, glucose and electrolytes, chest X-ray, or echocardiogram.

Subsequent Management

- Maintain normothermia with radiant warming. If necessary, wrap preterm infants' extremities in plastic wrap to prevent hypothermia. The larger body surface area and thin skin in the newborn may cause hypothermia to develop quickly.
- If the patient has moderate or severe hypoxic ischemic encephalopathy, therapeutic hypothermia may be appropriate. Request a critical care consultation for neonatal therapeutic hypothermia.
- After 10 minutes of *adequate* and uninterrupted resuscitation, discontinue efforts if no signs of life are present.

Risk Factors (for needing neonatal resuscitation)

- Approximately 10% of newborns do not start breathing without assistance. (Less than 1% need extensive measures for resuscitation.)
- Birth at less than <35 weeks postgestational age
- Oligohydramnios
- Chorioamnionitis or other infection
- Maternal hypertension

Risk Factors (continued)

- Fetal factors: Breech, shoulder dystocia, nonreassuring fetal heart rate, meconium in the amniotic fluid
- Use of opiates during otherwise normal labor
- Risk is *not* increased by assisted delivery (forceps or vacuum) or by maternal anesthesia (regional OR general). Risk is decreased by elective caesarean section.

Special Considerations

- The preductal SaO_2 in a neonate is not expected to reach infant levels until 10 minutes after birth. Beginning resuscitation with room air and increasing FiO_2 only if needed may lead to improved outcomes. Current guidelines state that not performing resuscitation is reasonable in patients with extreme prematurity (<23 weeks or birth weight <400 g), anencephaly, and chromosomal abnormalities that are incompatible with life (trisomy 13 or 18). Do not use naloxone to treat respiratory depression because it can cause sympathetic stimulation that can precipitate neonatal hemorrhagic stroke (intraventricular hemorrhage). If narcotic-induced respiratory depression is suspected, support ventilation with a bag and mask or endotracheal intubation (Table 9.3). If chronic opioid exposure has occurred, it may be necessary to gradually titrate a decreasing opioid dose over a period of days to weeks in order to avoid acute withdrawal.

Table 9.3 Endotracheal Tube Diameters and Lengths According to Gestation and Weight

50% weight by gestation		Tube diameter and length at the lip	
Gestation (weeks)	Body weight (kg)	Diameter (mm)	Length (cm)
23/24	0.5	2.5	6
26	0.75	2.5	6.5
27	1	2.5	7
30	1.5	2.5	7.5
33	2	2.5–3	8
35	2.5	3	8.5
37	3	3–3.5	9
40	3.5	3.5	9.5

Further Reading

American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care science: Part 15: Neonatal resuscitation. *Circulation*. 2010; 122: S909–S919.

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Pediatric Advanced Life Support: Outside the Operating Room

Definition

Guideline for cardiopulmonary resuscitation of a child or infant.

Presentation

- No response or minimal response to stimulation (tapping shoulder, calling name).

Respiratory Failure

- Apnea, cyanosis
- Impending respiratory failure can present with tachypnea, gasping, grunting, nasal flaring, paradoxical or diminished chest movement, or retractions.

Cardiac Arrest

- Pulseless: Electrocardiogram may show asystole, ventricular fibrillation, ventricular tachycardia, or may show a rhythm (pulseless electrical activity).
- Impending cardiac arrest associated with decompensated shock can present with tachycardia and hypotension in older children.

Pathophysiology

Lack of end-organ oxygenation, most commonly due to either respiratory failure (no oxygenation) or uncompensated shock (organs are not perfused with oxygen-rich blood), causing acidosis. Acidosis and hypoxia lead to myocardial ischemia and reduced cardiac contractility, eventually causing bradycardia, arrhythmias, and asystole.

DIFFERENTIAL DIAGNOSIS

- Look for and treat reversible causes. Hypoxia is the most common cause of cardiac arrest in children.
- “Hs and Ts”—a mnemonic for common causes—REVIEW EARLY IN RESUSCITATION.

- Hs: Hypoxia (airway obstruction, asphyxiation), H⁺ (acidosis), Hyper/Hypokalemia, Hypothermia, Hypovolemia (e.g., hemorrhage leading to shock), Hypoglycemia, Hypothermia
 - Ts: Trauma, Toxins, Tension pneumothorax (or hemothorax), Thrombosis (e.g., coronary artery, air embolus), Tamponade (cardiac)
- Arteriosclerotic myocardial infarction is an adult disease. Consider likely pediatric etiologies: anaphylaxis, congenital heart disease, foreign body aspiration, or ingestion of poisons (e.g., prescription medications!).

Immediate Management

- CAB: Circulation, airway, breathing
- Call for help.
- Call for a defibrillator/monitor.

For cardiac arrest:

- Circulation: Begin chest compressions at least 100 per minute. Minimum depth should be 1½ inches (4 cm) in infants and 2 inches (5 cm) in children, corresponding to one-third of the anteroposterior diameter of the chest. Allow full chest recoil between compressions. Continue compressions during other steps: No end-organ perfusion occurs when compressions are stopped (e.g., continue compressions while attaching defibrillator pads and establishing intravenous access).
- Breathing: Begin bag-mask ventilation if the patient is not intubated, looking for adequate chest rise. Single rescuers should give two breaths for every 30 chest compressions. If two rescuers are present, the second rescuer should give two breaths for every 15 compressions during a *brief* pause. Inspiratory time should be 1 second. Administer 100% FiO₂. Caution: Do not overventilate. If ventilation is difficult, consider two-person mask ventilation, with one provider using both hands on the mask, and the other provider squeezing the bag.

If the patient is intubated, ventilate without stopping chest compressions at a rate of 8–10 breaths/minute.

- Establish intravenous access.
- Attach defibrillator pads or paddles immediately, analyze for a rhythm, and shock if indicated (VF or VT). Many defibrillation units will automatically calculate the dose; if manual, use 2 J/kg for the first shock. Use the largest paddles or pads that can fit on the patient's chest without touching.

Immediate Management (*continued*)

- Resume chest compressions and ventilation immediately after each shock is given or if the defibrillator indicates “no shock.” Recheck the rhythm every five cycles (2 minutes) of CPR. The second and subsequent shocks should be 4 J/kg minimum (maximum dose is 10 J/kg or maximum dose for adults).
- Drug therapy: Administer epinephrine 0.01 mg/kg IV/IO every 3–5 minutes during resuscitation. Administer amiodarone 5 mg/kg IV/IO for ventricular fibrillation or ventricular tachycardia (may repeat twice if needed). Administer magnesium (25–50 mg/kg IV/IO: 25–50 mg/kg) for torsades de pointes (polymorphic VT with prolonged QT interval).
- Consider intubating the trachea if bag-mask ventilation is inadequate. Minimize interruptions in chest compressions. Attempt to measure end tidal CO₂, but absence may be caused by either esophageal intubation or lack of circulation (i.e., inadequate chest compressions). Note: Gastric inflation from overventilation can make ventilation difficult or impossible, especially in younger children. If ventilation becomes difficult, suction the stomach with an orogastric tube or a long suction catheter.
- **Call for ECMO early** if return of spontaneous circulation does not occur within the first 6 minutes of CPR

For respiratory arrest only:

- Bag-mask ventilation, give one breath every 3–5 seconds “squeeze-release-release.”
- Prepare for the possibility that cardiac arrest may be imminent.

For bradycardia (slow pulse causing decreased perfusion):

- Support the airway and breathing. Begin high-flow O₂. Initiate cardiopulmonary monitoring and establish intravenous or intraosseous access. Obtain a 12-lead ECG. Assess for and treat underlying causes (Hs and Ts). If decreased perfusion does not resolve by improving ventilation and oxygenation, begin CPR without delay.
- Drug therapy: After 2 minutes of chest compressions, give epinephrine IV/IO 10 MICROgrams/kg or endotracheally 100 MICROgrams/kg if needed. Administer atropine 20 MICROgrams/kg only if the primary cause is suspected to be AV block or vagal.
- Consider transthoracic or transvenous pacing, especially if a primary cardiac cause is suspected. Request an emergency consultation from a pediatric cardiologist.

Immediate Management (continued)

For tachycardia (with a pulse):

- Support airway, breathing, and circulation, administer high flow oxygen. Initiate cardiopulmonary monitoring, and establish intravenous or intraosseous access. Obtain a 12-lead ECG. Assess for and treat underlying causes (Hs and Ts).
- Narrow complex tachycardia (QRS <0.09 seconds):
 - Sinus tachycardia: look for and treat reversible causes.
 - *Stable* supraventricular tachycardia (SVT): Perform a vagal maneuver (place a bag of ice wrapped in a towel on the forehead in infants and toddlers, have older children blow through a straw, or perform a unilateral carotid sinus massage). Drug therapy (if vagal maneuvers ineffective): Administer adenosine 0.1 mg/kg IV/IO, flushed rapidly. In older children only, can considering administering verapamil 0.1–0.3 mg/kg.
- Unstable supraventricular tachycardia (SVT):
 - Sedate the patient (consider midazolam at least 0.1–0.2 mg/kg IV), administer synchronized cardioversion (administer 0.5–1 J/kg for the first shock, 2 J/kg for subsequent shocks if needed). If the rhythm does not convert or SVT occurs again quickly, administer amiodarone 5 mg/kg IV/IO or procainamide then give a third shock.
- Wide complex tachycardia (QRS >0.09 seconds):
 - This may be ventricular tachycardia. If unstable, administer synchronized cardioversion (0.5–1 J/kg for the first shock, 2 J/kg for subsequent shocks if needed). If stable, request an emergency consultation from a pediatric cardiologist.

Diagnostic Studies

- Do not interrupt CPR unnecessarily for blood draws or delay treatment of reversible causes while waiting for laboratory values. Consider arterial blood gas analysis, serum electrolytes, glucose, and calcium, as well as a carboxyhemoglobin level and toxicology screen. If a toxic ingestion is suspected, contact a toxicologist or poison control!
- Echocardiography can identify cardiac tamponade, massive embolus, poor cardiac contraction, and decreased preload. Do not delay treatment for an echocardiogram if one of these causes is suspected.

Subsequent Management (After Successful Resuscitation from Cardiac Arrest)

- Decrease the FiO_2 while maintaining a $\text{SaO}_2 >94\%$.

- Monitor for adequate perfusion and O_2 delivery. Over time, acid-base status will normalize and lactate will decrease if resuscitation is successful.
- Consider administering a vasopressor (e.g., dopamine) for cardiac output caused by myocardial dysfunction.
- Therapeutic hypothermia (core temperature 32°C to 34°C for 12–24 hours) may improve neurologic outcome in children who remain unconscious following successful resuscitation.
- Treat hyperthermia (core temperature $>38^\circ\text{C}$) to improve neurologic outcome.

Risk Factors

- Insufficient supervision leading to trauma or drowning
- Congenital disease: Down syndrome (bradycardia and cardiac arrest can occur even in the absence of structural heart disease), long QT syndrome, family history of sudden cardiac arrest
- Children who were born prematurely

Special Considerations

- The American Heart Association changed its guidelines for the management of cardiac arrest in children and adults in 2010 to prioritize chest compressions. The mnemonic has changed from “ABC” to “CAB”—circulation, airway, breathing. Although the most likely cause of cardiac arrest in children is a respiratory event, mobilizing supplies for airway management (bag, mask) typically requires at least several seconds, whereas chest compressions can be started immediately.
- Minimize interruptions in chest compressions, even when checking the pulse or securing the airway. It is difficult to maintain high-quality compressions for long; the rescuer performing compressions should rotate every 2 minutes.
- Patients receiving CPR are commonly overventilated, which decreases the likelihood of successful resuscitation. Overinflation of the lungs decreases venous return, decreasing the amount of blood that the heart can then pump to the brain, end-organs, and coronary arteries. Inflation of the stomach during overventilation also increases the risk of aspiration of stomach contents into the lungs.
- $ETCO_2$ and arterial blood pressure indicate adequate chest compressions and return of spontaneous circulation. $ETCO_2$ consistently >10 – 15 mm Hg indicates high-quality CPR (adequate chest compressions and recoil, absence of

overventilation). A sudden and sustained increase in ETCO_2 may indicate the return of spontaneous circulation. Caution: For 60–120 seconds following intravenous vasoconstrictors (e.g., epinephrine, vasopressin), ETCO_2 may decrease due to the concomitant decrease in pulmonary blood flow; this is usually an artifact.

- Pulse oximetry is not accurate in patients with decreased perfusion, and may appear normal in patients with carbon monoxide poisoning.
- If intravenous access cannot be secured, obtain intraosseus access promptly. Intraosseus access can be used to administer all medications, fluids, and blood products. Intraosseus administration is preferable to endotracheal administration of medications, although if absolutely necessary, naloxone, epinephrine, atropine, and lidocaine (NEAL) can be administered through the endotracheal tube. The endotracheal dose is two to three times the intravenous dose for naloxone, atropine, and lidocaine, and 10 times the intravenous dose for epinephrine.
- Parents should be given the option of being present during the resuscitation, Class I evidence shows that being there will help families to later process this traumatic experience.

Further Reading

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care: Part 13: Pediatric basic life support. *Circulation*. 2010; 122: S862–S875.

American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care: Part 14: Pediatric advanced life support. *Circulation*. 2010; 126: e1361.

Stridor, Postextubation

Definition

Upper airway narrowing (proximal trachea or above) that reduces airflow and may lead to respiratory failure.

Presentation

- Typically seen after extubation, especially if the endotracheal tube was too large (no leak at a pressure of 20–25 mm Hg) or after prolonged intubation.
- A high-pitched, “squeaking” sound may be heard on auscultation over the anterior neck, and will be associated with tachypnea,

hypoxemia, and hypercarbia. Sternal retractions and nasal flaring are caused by increased work of breathing.

Pathophysiology

Stridor is caused by fast, turbulent airflow in the oropharynx, larynx, or upper trachea. It occurs during inspiration because abnormal or inflamed upper airway tissue is pulled inward, generating turbulence and noise. Poiseuille's law dictates that airway obstruction increases 16 times for each 50% reduction in the airway radius.

Immediate Management

- Administer humidified 100% O₂ via blow-by or face mask, as tolerated.
- Administer nebulized racemic epinephrine 0.5 mL of 2.25% solution, diluted in 2 cc normal saline, 0.25 mL for infants.
- Administer dexamethasone 0.5–1 mg/kg (airway dose) if not already given during the surgical procedure. This will decrease airway inflammation after 4–6 hours.
- In severe cases: Continuous positive airway pressure, consider reintubation (may need a smaller endotracheal tube).
- In severe cases: Consider administering helium-oxygen mixture (Heliox) to decrease turbulent airflow.

275

DIFFERENTIAL DIAGNOSIS

- Bronchospasm
- Laryngospasm
- Acute allergic reaction (often accompanied by rash and hypotension)
- Vocal cord dysfunction caused by recurrent laryngeal nerve injury
- Foreign body aspiration (e.g., tooth dislodged during laryngoscopy)
- Tracheomalacia or laryngomalacia (prematurity, prior tracheostomy) Caution: Sedation will exacerbate airway obstruction in these patients.

Diagnostic Studies

- Clinical diagnosis

Subsequent Management

- Reintubation and/or pediatric intensive care unit management may be needed for severe cases.

Risk Factor

- Younger age

Further Reading

da Silva PS, Fonseca MC, Iglesias SB, Junior EL, de Aguiar VE, de Carvalho WB. Nebulized 0.5, 2.5 and 5 ml L-epinephrine for post-extubation stridor in children: a prospective, randomized, double-blind clinical trial. *Int Care Med*. 2012; 38(2): 286–293.

Trauma

Definition

Mechanical injury, often involving multiple organ systems, and which may cause rapid deterioration. Associated injuries, sometimes remote in location from the primary injury, can increase morbidity and mortality. Requires simultaneous evaluation and implementation utilizing advanced cardiac life support—ACLS (to maintain end-organ perfusion) and advanced trauma life support—ATLS (for injury management).

Presentation

276 Advanced Trauma Life Support (ATLS) evaluation begins with the primary survey (ABCDE—Airway and cervical Spine, Breathing and ventilation, Circulation and hemorrhage management, Disability and neurologic status, Exposure and environment). Ignoring or delaying any of these components may cause serious morbidity; therefore, these steps are taken simultaneously, guided by a team leader. This is followed by the secondary survey, which is an assessment from head-to-toe and re-evaluation of vital signs. The components of the primary survey must be constantly re-assessed due to the potential for a worsening of the patient's condition.

Airway and Cervical Spine

- If talking or crying, the patient is able to exchange air. Stridor may indicate imminent airway obstruction. Chest rise can be misleading, especially in children, as it can be seen in the absence of air movement (i.e., if patient has respiratory effort but complete airway obstruction).
- Unconscious patients are at high risk for aspiration and airway obstruction.
- Obtunded or unconscious patients, or those with a distracting injury, cannot be evaluated clinically for a cervical spine injury.

Breathing and Ventilation

- Hypoxia and cyanosis (from hypopnea, pulmonary contusions, flail chest, pneumothorax, hemothorax, or aspiration)
- Tachypnea and respiratory distress, including paradoxical chest movement, retractions, nasal flaring, gasping, grunting

- Imminent airway loss and need for intubation if the patient is hypopnic, if mask ventilation is impossible, or if the patient has signs of respiratory distress, a C1/C2 injury, a Glasgow Coma Scale (GCS) <9, penetrating neck injury, is hemodynamically unstable, or has a major pulmonary or chest wall injury.
- Visible tracheal deviation or penetrating injury. Subcutaneous emphysema or unilateral breath sounds (pneumothorax, hemothorax) may also be present.

Circulation and Hemorrhage Management

- In children, blood pressure will not decrease until >25%–30% of blood volume has been lost. The first sign of hypovolemia in children is an increase in heart rate.
- Can have hidden blood loss into the intrathoracic, intraperitoneal, or retroperitoneal spaces, or into the pelvis or long bones.

Disability

- Assess neurologic status with the mnemonic AVPU—alert, verbal stimuli response, painful stimuli response, unresponsive.

Exposure and Environment

- Examine completely by undressing the patient.
- Take steps to prevent hypothermia.

Secondary Survey

- AMPLE—Allergies, Medications, Past medical problems and surgery, Last Meal, Events related to the injury
- Glasgow Coma Scale
- Head-to-toe evaluation

Pathophysiology

Due to a child's small size, internal organ and multiple system injuries are common in major trauma, as force is transmitted quickly through the more elastic skeleton and connective tissue. Hemorrhage can cause rapid decompensation because of the smaller total blood volume. Hypothermia occurs quickly in these patients with a large body surface area relative to total body mass. Coagulopathy is a concern in patients who are transfused more than one blood volume.

DIFFERENTIAL DIAGNOSIS

- Toxins (ingestion of household chemicals, unsecured medications, or drug or alcohol abuse).
- Child abuse (story inconsistent with injuries, multiple healing fractures of varying age, detached retina).

Immediate Management

Airway and Cervical Spine

- In an awake patient, provide supplemental oxygen by face mask or nasal cannula as needed to maintain $\text{SaO}_2 > 90\%$.
- Use of a correctly sized cervical spinal collar is required after blunt trauma injuries for immobilization if the C-spine cannot be immediately cleared. Clearance requires five clinical criteria: no midline cervical tenderness, no focal neurologic deficit, normal alertness, no intoxication, and no painful distracting injury.
- If the patient appears unconscious or has a severely altered mental status, keep the C-spine immobile while performing a chin lift and jaw thrust and suctioning any obstructing airway secretions and blood. If the patient “wakes up” when suctioned and can protect his airway, intubation might not be needed.
- Intubate if minimal consciousness, severe injury, or imminent loss of airway. If the patient was intubated pre-hospital, confirm proper ETT size and placement. Utilize a “rapid-sequence” induction, with the c-collar removed and one person keeping the C-spine immobile throughout the time the collar is off with manual in-line stabilization.
- Have suction and emergency airway equipment immediately available. Visualization of the airway may be difficult due to distortion and bleeding. A supraglottic airway, such as a LMA, may be life-saving in cases of difficult ventilation and difficult intubation. If a difficult airway is anticipated, have equipment and a surgeon qualified to perform a surgical airway immediately available.

Breathing and Ventilation

- Dependent on injury: pneumothorax may require immediate needle decompression and a chest tube; lung injury may require immediate operative management.

Circulation and Hemorrhage Management

- Establish two large-bore intravenous lines in major trauma. In infants and small children, can often only obtain 22–24g IVs. Consider intraosseous access if intravenous access is not feasible.
- Assess and control bleeding, external and internal. Emergency surgery may be required.

Immediate Management (continued)

- If the patient is hypotensive, begin fluid resuscitation with normal saline or lactated Ringers; administer a bolus of 20 mL/kg. May need to be repeated.
- For major bleeding, begin transfusion with blood products as early as possible. May need to activate the massive transfusion protocol.
- In patients with severe injury, consider invasive monitoring (intra-arterial catheter, central venous catheter), orogastric tube, and bladder catheter.

Disability and Neurologic Status

- Patients with altered neurologic status may have a brain injury.

Exposure and Environment

- After major trauma, remove all clothing to assess for associated injuries.
- Monitor temperature and maintain normothermia by warming the room, utilizing forced air warming devices, and warming intravenous fluids and blood products.

Diagnostic Studies

- Chest radiograph
- If the patient is stable, advanced imaging such as a CT scan may be indicated.
- Serial hemoglobin and hematocrit, toxicology screen, electrolytes, coagulation panel. A thromboelastogram may be useful to quickly obtain information about coagulation and qualitative platelet function.

Subsequent Management

- In patients with acute head injury: avoid steroids for increased intracranial pressure, avoid prolonged hyperventilation, do not routinely give anticonvulsants to patients who have not had a seizure.
- Maintain oxygenation and perfusion. (Cerebral and spinal cord perfusion pressures are particularly important if these structures are injured.)
- Only use glucose-containing intravenous fluids if patient is hypoglycemic, especially for traumatic brain injury or spinal cord injuries.
- Do not give steroids for spinal cord injury (Level I evidence in adults), pediatric evidence pending.
- Manage acute pain with opioids, nonsteroidal anti-inflammatory drugs (NSAIDs), acetaminophen, low-dose ketamine infusions for NMDA blockade (e.g., 0.1 mg/kg/h), or regional

anesthetic techniques (particularly for orthopedic injuries). Patient-controlled analgesia (PCA) may be appropriate for children 5 years or older.

Risk Factors

- Riding in the car without a car seat or safety belt (as appropriate for age)
- Not wearing a helmet for high-speed activities (e.g., bike riding, skiing, ATV riding)
- Child abuse

Prevention

Prenatal, postpartum, and infant/toddler home visits by nurses for disadvantaged children through the nurse–family home partnership significantly decrease mortality from both accidental and intentional injury.

Special Considerations

- Patients suffering from massive hemorrhage quickly become coagulopathic and acidotic. These factors can be made worse with the administration of large amounts of crystalloid fluid and packed red blood cells, which can cause hemodilution and hypothermia. Increasing evidence has been found for “damage-control resuscitation,” which involves early blood product administration (platelets, fresh-frozen plasma, and PRBCs in a 1:1:1 ratio) and limited crystalloid infusion, as well as accepting a slightly lower blood pressure in settings of uncontrolled hemorrhage.

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Chapter 10

Miscellaneous Problems

Sara E. Neves and Keith J. Ruskin

Acute Transfusion Reaction	282
Bone Cement Implantation Syndrome	284
Burns	285
Dental Trauma and Oral Injury	287
Drug Extravasation	290
Intra-arterial Injection	292
Magnetic Resonance Imaging Emergencies	294
Occupational Exposure	296
Operating Room Fire	298

Acute Transfusion Reaction

Definition

Intravascular hemolysis of red blood cells caused by recipient antibody binding to donor antigens, activating complement and causing hemolysis. Usually occurs in response to a major ABO-type mismatch, but can also occur with other red blood cell (RBC) antigens in patients with prior transfusion and alloimmunization.

Presentation

- Variable symptoms, many of which are masked by anesthesia.
- Hypotension
- Hemoglobinuria
- Bleeding diathesis
- An awake patient may complain of nausea, fever, chills, and chest and flank pain.

Pathophysiology

Antibodies in the recipient plasma bind to donor RBC antigens and activate the complement pathway, resulting in intravascular and extravascular hemolysis and the release of bradykinin and histamine. Severe transfusion reactions result in renal failure (likely due to hemoglobin precipitating in the distal tubules) and disseminated intravascular coagulation (DIC), which commonly occurs when RBC products are released and activate the intrinsic system of coagulation.

DIFFERENTIAL DIAGNOSIS

- Sepsis
- Delayed transfusion reaction (transfusions within 2–21 days)
- Febrile transfusion reaction (direct antiglobin test)

Immediate Management

- Stop the transfusion.
- Maintain urine output at 75–100 cc/hour with generous intravenous (IV) fluid administration.
- Consider mannitol 12.5–50 g IV.
- Consider furosemide 20–40 mg IV.
- Alkalinize the urine to pH of 8 with sodium bicarbonate (0.5–1 mEq/kg, then additional doses as necessary to achieve urine pH of 8).
- Maintain blood pressure as needed.

Diagnostic Studies

- Send suspected unit to blood bank with another sample of patient's blood for repeat cross-match.
- Send a blood sample to blood bank for direct antiglobulin test and hemoglobinemia.
- Send urine for hemoglobinuria.

Subsequent Management

- Maintain normotension (using fluids and vasoactive drugs as necessary) to ensure adequate renal blood flow.
- Send urine and serum samples for hemoglobin concentration.
- Send blood samples for platelet count, partial thromboplastin time, and fibrinogen level.
- Consider a hematology consult.

Risk Factors

- The majority of transfusion reactions are due to ABO incompatibility. Most errors occur *after* the blood products have left the blood bank and are committed by physicians and nurses.
- Rushed or incomplete check-in, especially during rapid blood loss or trauma surgery.
- Labeling errors: Check the patient's unit number and blood serial number carefully.

Prevention

Use extra caution when administering blood products.

Special Considerations

Delayed hemolytic transfusion reactions may occur between 2 days and 3 weeks after administration of blood products, and usually manifest as a drop in hematocrit. They are difficult to prevent, since they require very low levels of antibody that may be undetectable. Severe hemolytic reactions are fatal in 20%–60% of patients.

Further Reading

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Bone Cement Implantation Syndrome

Definition

Hypoxia, hypotension, cardiac dysrhythmias, or increased pulmonary vascular resistance following methyl methacrylate cement implantation.

Presentation

- Decreasing end-tidal carbon dioxide (ETCO₂) in ventilated patients
- Dyspnea and/or altered mental status in patients under regional anesthesia
- Cardiac dysrhythmias or cardiac arrest may occur.

Pathophysiology

The pathophysiology of bone cement implantation syndrome (BCIS) is unknown. Proposed mechanisms include embolic showers causing either a direct mechanical effect or release of vasoactive or proinflammatory mediators. Complement activation has also been proposed as a mechanism.

DIFFERENTIAL DIAGNOSIS

- Anaphylaxis
- Massive pulmonary thromboembolus (prolonged hypoxemia)
- Myocardial infarction or cardiac failure (electrocardiogram changes, prolonged hypotension, pulmonary edema, minimal response to fluids or vasopressors)

Immediate Management

- Increase FiO₂ to 100%.
- Initiate aggressive resuscitation with IV fluids.
- Support blood pressure with ephedrine (5 mg IV) or phenylephrine (100-mcg IV) boluses. If refractory, consider phenylephrine or epinephrine infusion.

Diagnostic Studies

- Bone cement implantation syndrome is a clinical syndrome without specific confirmatory diagnostic studies.
- Transesophageal echocardiography or precordial Doppler ultrasonography may reveal the presence of emboli.

Subsequent Management

- Advise the surgical team of the event and make a decision as to whether to proceed if a second joint replacement is planned.

- Bone cement implantation syndrome is usually transient and resolves spontaneously.
- Prolonged episodes may occur and should be treated as right ventricular failure. In this case, consider placement of a central venous catheter for monitoring and administration of vasoactive drugs.

Risk Factors

- Rare in healthy patients
- More common in elderly or debilitated patients
- May be associated with surgical technique

Prevention

The severity of BCIS may be reduced by generous fluid administration, increased vigilance during and immediately after prosthesis implantation, and consideration of invasive blood pressure monitoring in high-risk patients.

Special Considerations

Intravascular emboli of air, bone marrow, or fat can be seen on transesophageal echocardiography during implantation of orthopedic prostheses. Some of these patients develop transient hypotension or hypoxemia.

Further Reading

Donaldson AJ, Thomson HE, Harper NJ, Kenny NW. Bone cement implantation syndrome. *Br J Anaesth*. 2009; 102(1): 12–22.

Burns

Definition

First-degree burns involve epidermis and upper dermis and heal spontaneously. Second-degree burns involve the deep dermis and require excision and grafting. Third-degree burns involve complete destruction of the dermis and must be excised and grafted. Fourth-degree burns involve muscle, fascia, and bone.

Presentation

- Thermal trauma after exposure to flames in an enclosed space
- Thermal trauma after airplane, motor vehicle, or industrial accidents
- Chemical burns after industrial accidents

- Partial-thickness burns are red, blanch when touched, and heal spontaneously. Full-thickness burns do not blanch and are insensate.
- Airway injury from smoke inhalation present with dyspnea and airway obstruction (airway injury may not be immediately apparent)

Pathophysiology

Severe burns cause multiple systemic reactions, including release of interleukins and tumor necrosis factor, resulting in immunosuppression, sepsis, multiple organ failure, and protein catabolism. Hypoxemia may result from lung injury, atelectasis, and airway edema. Extensive fluid loss from the injury and massive fluid shifts may cause hypovolemic shock.

Immediate Management

- Immediately administer 100% O₂ by face mask in patients with a patent airway.
- Secure the airway with an endotracheal tube. Awake fiberoptic intubation with topical anesthesia is preferred in patients with severe facial or airway injury, but other techniques may be considered.
- After intubation, maintain a high FiO₂ due to the risk of CO toxicity.
- Begin aggressive fluid resuscitation in patients with burns >15% total body surface area (TBSA). Crystalloid resuscitation is preferred in the first 24 hours following burn injury. Estimate requirements according to the Parkland formula:

$$\text{Fluid requirements} = \text{TBSA burned (\%)} \times \text{Wt (kg)} \times 4 \text{ mL}$$

- Administer one-half of the total requirement in first 8 hours; give the second half over the next 16 hours.
- Fluid management is guided by urine output, central venous pressure (CVP), or pulmonary artery pressures.
- If cyanide toxicity is suspected, administer sodium thiosulfate, sodium nitrate 3% solution, and hydroxycobalamin.
- If a chemical burn is suspected, use caution to prevent contamination of unit or staff.

Diagnostic Studies

- Surface area can be estimated by the Rule of 9s: Head 9%; each upper extremity 9%; each lower extremity 18%; torso front and back 18% each.
- Blood electrolytes

- Arterial blood gas, including co-oximetry to determine carboxyhemoglobin level
- Lactic acid level (lactic acidosis may indicate cyanide poisoning from burning plastics).

Subsequent Management

- Maintain normothermia. Use warming blankets, forced-air warmers, fluid warmers as necessary. Keep the room temperature as high as possible.
- Use topical antibiotics to prevent infection.
- Consider hyperbaric oxygen therapy if the patient is stable, a pressure chamber is available, and severe CO poisoning is suspected.

Risk Factors

Fires in the operating room due to electrocautery or lasers. “Fire resistant” plastic drapes will burn in the presence of O₂ and release toxic smoke.

Prevention

See Operating Room Fires (page 298).

Special Considerations

- Full-thickness burns appear white, waxy, or leatherlike and may be confused with unburned skin. Full thickness burns do not bleed.
- Succinylcholine is generally safe to use within the first few hours after a burn, but after that must be avoided for 12 months after the burn injury.
- Resistance to nondepolarizing neuromuscular blocking agents may occur for up to 10 weeks postinjury.

Further Reading

Hettiaratchy S, Papini R. Initial management of a major burn: I—overview. *BMJ*. 2004; 328(7455): 1555–1557.

Hettiaratchy S, Papini R. Initial management of a major burn: II—assessment and resuscitation. *BMJ*. 2004; 329(7457): 101–103.

Dental Trauma and Oral Injury

Definition

Lacerations, abrasions, and perforation of oral tissues or mucosa, and fracture or avulsion of teeth.

Presentation

- Occurs most often during airway management: placement/removal of oral airways, laryngeal mask airways (LMAs), endotracheal tubes, laryngoscope blades.
- Glidescope intubation with a rigid stylet can cause laceration or perforation of soft palate and other structures due to poor visibility.
- Procedures such as endoscopy, bronchoscopy, and maxillofacial or otorhinolaryngeal surgery may result in non-anesthesia related oral/dental injury.
- Shivering can result in masseter muscle spasm, which can damage teeth.
- Injury may be recognized immediately as it occurs, present with symptoms of aspiration during surgery, or only be noticed in the postoperative period in the postanesthesia care unit or later by the patient or his or her family members.

Pathophysiology

Patients with pre-existing dental conditions are more likely to have dental damage during surgery and maxillary incisor teeth are at greatest risk for trauma during anesthesia. Poor oral hygiene compromises the integrity of the tooth, weakening tooth structure and making the tooth more prone to fracture. Periodontal disease makes teeth more susceptible to avulsion because it affects the gingiva, bone, and ligaments that keep the teeth in place. Aggressive suctioning, forceful insertion or removal of oral airways, and accidental contact of the laryngoscope blade with the teeth can also cause damage such as breaking or dislodging of teeth and dental restorations (e.g., adhesive bondings, caps/crowns, bridges, and dentures). Such injuries are more likely to occur in patients who are difficult to intubate. Aspiration of teeth, blood, tissue, or hardware can cause significant respiratory injury, and sometimes can cause GI injury when ingested. Dental damage can incur significant cost as well as emotional distress for the patient.

Immediate Management

- If the injury occurs during airway management, secure the airway first before examining the teeth for injury.
- After the airway is secure, examine oral cavity carefully. If a tooth has been avulsed, ascertain that it is intact with no pieces missing. If a tooth is fractured or hardware is broken, look for any missing pieces.
- If any teeth or hardware cannot be accounted for, obtain an intraoperative chest X-ray to exclude aspiration or ingestion.
- If a tooth has been aspirated or ingested, request an intraoperative ear-nose-throat (ENT) or pulmonary medicine

Immediate Management (*continued*)

consult (to remove an aspirated tooth) or a GI consult (to determine if endoscopy is necessary). Consider aborting the surgery if it cannot be retrieved.

- If a soft tissue injury has occurred, carefully examine the area, clean with normal saline. If necessary, request an intraoperative ENT consult.
- For an avulsed tooth, place the tooth and any pieces in normal saline and request a dental consult. Do not attempt to clean or scrub the tooth with alcohol or other cleansers and try to avoid holding tooth by the root because this may damage ligaments needed for reattachment.

Diagnostic Studies

- Chest X-ray to determine location of missing teeth or hardware.

Subsequent Management

- Document injuries carefully and thoroughly.
- Discuss with surgical team the presence and cause of any injury prior to discussion with patient and family.
- Have a frank and thorough discussion with the patient about the injury, and involve risk management personnel according to the hospital protocols.

Risk Factors

- Pre-existing dental disease and poor oral hygiene.
- Hardware or prosthetics, which are not as strong as original dentition.
- Emergency airway management
- Difficult airway
- Pediatric patients between the ages of 5–12 who have both primary and permanent teeth.

Prevention

- Include a preoperative dental history and examination as part of airway examination, paying particular attention to the maxillary anterior teeth, because dental treatment of these teeth may not be readily apparent and these teeth are at greatest risk. In a high-risk patient, a more thorough physical examination of the gingiva and teeth may offer insight into the likelihood of dental trauma. Document prior dental damage and existing restorations precisely. Having a discussion with the patient specifically addressing the risk of oral injury and dental trauma

preoperatively may prevent emotional distress and legal action later. If necessary, obtain preoperative consultation by a dentist to extract any loose or infected teeth.

- Use care when manipulating airway devices to avoid pinching or lacerating lips or soft tissue. If a videolaryngoscopy device is used for intubation, maintain direct visualization of endotracheal tube as it enters the oral cavity and the back of the pharynx until tube is visible on video screen. Avoid blind, forceful insertion of the blade or endotracheal tube.
- Maintaining normothermia can prevent masseter muscle spasm due to shivering, and the use of soft bite blocks can prevent dental trauma resulting from clenching down on oral airways, LMAs, and endotracheal tubes. Bite blocks also prevent the need to forcibly pull the airway device out when it is no longer needed.

Special Considerations

- Patients with poor dentition may have an unrecognized abscess or otherwise prove an infection risk for surgery, especially in immunosuppressed patients or for procedures in which hardware is being implanted.
- Time elapsed to reimplantation after avulsion of a tooth is the main determinant of success. It is especially important that the avulsed tooth be kept moist in saline to improve likelihood of reattachment later. A dental consultation should be requested as soon as possible after the injury.

Further Reading

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Drug Extravasation

Definition

Unintentional injection of drugs or fluids into the subcutaneous tissue or perivascular space.

Presentation

- Pain on injection or during infusion of fluids.
- Discomfort, swelling, or hyperemia at the site of the catheter.
- Paresthesias or local induration of the skin (late signs).

- Severe cases: Compartment syndrome; muscle, tendon, or nerve injury.

Pathophysiology

Tissue injury occurs for a variety of reasons, including hydrostatic pressure, fluid osmolality or cytotoxicity, vasoconstriction.

DIFFERENTIAL DIAGNOSIS

Intra-arterial injection (severe burning pain, discoloration, or absence of pulse distal to injection site)

Immediate Management

- There is no definitive treatment for extravasation injury.
- Stop drug injection or fluid administration if the patient complains of severe pain or signs of extravasation are noted.
- Consider vascular or plastic surgery consultation.
- Specific treatment depends on the extravasated substance.
- In the case of vesicants (e.g., adriamycin), stab incisions and flushing with 500 mL of normal saline has been recommended.
- Extravasation of vasopressors has been treated with phentolamine infiltration.

291

Diagnostic Studies

- Clinical diagnosis: No diagnostic studies are necessary.
- Subsequent management
- Document the injury in the patient's chart.
- Observe the site carefully for at least several days.

Risk Factors

- Location of catheter
- High infusion pressure
- Multiple punctures of the same vein
- Access sites in close proximity to tendons, nerves, or arteries

Prevention

Avoid placing IV catheters over joints (e.g., antecubital fossa). Whenever possible, place IVs where they can be visually inspected throughout the surgical procedure. Avoid using "positional" IVs. If there is any doubt, administer a small test dose of drug first.

Special Considerations

- Although elevating the extremity and applying warm or cold compresses are commonly recommended, there is little evidence to support these practices. Extravasation can occur even when

a central venous catheter is used because the proximal port may exit the vessel if the catheter is withdrawn even a few centimeters. Therefore, vassicants should be given through the distal lumen.

Further Reading

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Intra-arterial Injection

Definition

Unintentional injection of drugs or fluids into an artery, usually by injection into an indwelling intra-arterial catheter, or after accidentally inserting an intravenous catheter into an artery.

Presentation

- Pain on injection or during infusion of fluids, possibly in the distribution of the vessel.
- Anesthesia or muscle weakness may occur distal to the injection site in an awake patient.
- Skin pallor or cyanosis distal to the injection site.

Pathophysiology

Pallor, ischemia, and pain may occur due to vasospasm, chemical arteritis, or drug-induced tissue injury.

DIFFERENTIAL DIAGNOSIS

- Drug extravasation (discomfort, swelling or hyperemia at the site of the catheter; late signs include paresthesias or local induration of the skin)
- Some drugs (e.g., propofol) cause pain on injection. This will not be associated with distal blanching or mottling of the skin.

Immediate Management

- Stop drug injection or fluid administration if the patient complains of pain or distal pallor is noted.
- Leave the catheter in place for subsequent diagnostic studies and treatment. Begin a slow infusion of isotonic saline solution if the catheter can be flushed and there is no clot.

Immediate Management (*continued*)

- Consider anticoagulation with intravenous heparin infusion.
- Consider intra-arterial lidocaine 2 mg/kg and papaverine (30 mg).

Diagnostic Studies

- Clinical diagnosis
- Transduce the catheter. High pressures or an arterial waveform implies that the catheter is intra-arterial. If the patient has an arteriovenous fistula, this test is nondiagnostic.

Subsequent Management

- Document the injury in the patient's chart.
- Extremity should be elevated to decrease edema and reduce the risk of compartment syndrome.
- Consider sympathectomy with a stellate ganglion block or continuous brachial plexus block (if catheter is located in upper extremity).
- Consider intra-arterial thrombolytic injection.
- Observe the site carefully for at least several days.

Risk Factors

- Morbid obesity
- Darkly pigmented skin
- Thoracic outlet syndrome (pulse decreases with internal rotation of the arm)
- Pre-existing vascular anomaly

Prevention

- Always verify that drugs are being injected into the correct tubing in a patient with an arterial catheter.
- Never assume that an indwelling catheter is in the correct location.
- When inserting an intravenous catheter, the tourniquet should not be so tight as to occlude arterial blood flow.
- Before injecting a drug, carefully observe IV tubing for backflow of blood.

Special Considerations

Although many treatment strategies have been recommended, none has been definitively proved to work. All recommendations are based on individual case reports or small series. Vigilance, immediate recognition of the problem, discontinuation of the irritant, and rapid initiation of treatment offer the best chance of a good outcome.

Further Reading

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Magnetic Resonance Imaging Emergencies

Definition

Anesthesia is commonly required for magnetic resonance imaging (MRI), whether for pediatric patients or adult patients unable to remain stationary for the required amount of time. Increasingly, MRI-guided procedures are being performed that require the anesthesiologist to work in this high-risk environment.

Presentation

- Injuries to staff or the patient can occur when magnetic resonance unsafe equipment is brought into the magnetic field.
- Respiratory failure or cardiac arrest during the scan when there is limited access to the patient can be life threatening.
- Burns can occur during a scan due to conductive materials present on the patient's skin.
- Quenching the magnet results in the release of heat as well as boiling off of the cryogenic fluid; usually helium. If the exhaust mechanism malfunctions, the gas produced poses an asphyxiation risk to anyone in the room.

Pathophysiology

Magnetic resonance imaging relies on the energy difference of the magnetic field of the scanner and the tiny magnetic fields of the atomic nuclei in every cell in the body. The scanner applies an oscillating magnetic field to excite the nuclei, and then as they relax back to their original state a receiving coil detects that energy difference. These data are then converted to usable clinical information as the MRI image. Typical scanners have magnetic fields between 0.5 and 3 Tesla; the areas around the scanner are demarcated to indicate safe limits and are described in Gauss (G). Any magnetic resonance unsafe equipment or personnel not cleared for MRI should remain outside the 5G line. The magnetic field of the scanner is always present, even when not actively scanning; therefore, a high degree of vigilance is required at all times.

Immediate Management

- Call for help.
- Quickly verify one's own magnetic resonance safety before approaching a patient in the scanner (no scissors in pocket, for example).
- Determine if there is time for a controlled shut-down of the scanner, or if an emergency quench is necessary.
- Despite the unusual environment, refer to basic training: ABCs, presence of suction prior to intubation, etc.
- If the magnet must be quenched, quickly locate source of oxygen for oneself as well as for the patient.
- For any burns related to MRI scanner, see section on Burns.

Diagnostic Studies

- Typically there are no diagnostic studies other than those needed for pre-MRI evaluation (computed tomography or X-ray if there is a question of metallic foreign body) or those needed for a patient who has a medical emergency during a scan.

Risk Factors

- Staff that is not trained in the risks of working in an MRI environment.
- Magnetic resonance—unsafe or conditional equipment left in an inappropriate area.
- Incomplete or erroneous evaluation of the patient's safety for MRI.
- Lack of preparation for working in an MR environment.

Prevention

- All staff, including attending physician, residents, medical students, and cleaning personnel, should be trained in MRI safety before entering an MRI suite.
- Patients should be evaluated carefully before receiving MRI scan to ensure they have no ferromagnetic foreign bodies. If this information cannot be verified, alternative imaging techniques should be considered.
- All equipment should be checked for MR compatibility prior to start of case.
- An emergency plan should be formulated before starting the scan. The location and availability of emergency equipment should be verified and forms of communication (intercom, phone) should be reviewed.
- Do not quench the magnet unless a person is in immediate danger.

Special Considerations

The MRI scanner is associated with significant acoustic noise. Although the noise level does not present an emergency, it is at a level higher than the noise level designated as safe by the Occupational Safety and Health Administration. Patients (whether awake or anesthetized) and providers should always wear ear protection during a scan. It may be difficult to hear alarms or to communicate with other personnel while a scan is in progress.

Further Reading

Reddy U, White MJ, Wilson SR. Anaesthesia for magnetic resonance imaging. *Contin Educ Anaesth Crit Care Pain*. 2012; doi:10.1093/bjaceaccp/mks002

Occupational Exposure

Definition

Inoculation of a health care worker (HCW) with infectious blood or body fluids by puncturing the skin with a contaminated object (e.g., needle) or splashing fluids into exposed mucosa.

Presentation

- Injury with a needle or other sharp object.
- Blood or body fluids splashed into the eyes, mouth, or an open wound.

Pathophysiology

Exposure to blood-borne pathogens results in infection of a health care worker. Twenty-five percent of all percutaneous sharps injuries among health care workers occur in OR personnel.

Immediate Management

- Wash the wound liberally with soap and water. Use of antiseptics is not contraindicated, but there is no evidence that use will reduce risk of infection.
- If exposure was through exposed mucous membranes, they should be irrigated copiously with normal saline.
- All health care institutions have an occupational exposure protocol; this protocol should be followed.
- Federal (US) and state reporting requirements also must be followed.
- Postexposure prophylaxis depends on the type of suspected pathogen.

Diagnostic Studies

- The patient should be tested for the presence of blood borne pathogens hepatitis B surface antigen (HBsAg), hepatitis C virus antibody (anti-HCV), and HIV antibody if his or her status is unknown. Testing usually requires patient consent.
- If exposure to HCV is suspected, the health care worker should undergo baseline testing for anti-HCV and alanine aminotransferase with follow-up in 4–6 months.
- If exposure to HIV is suspected, the health care worker should undergo baseline testing for HIV antibody and further testing at 6–12 weeks and 6 months.

Subsequent Management

- If the exposure was to hepatitis B and the HCW is susceptible, hepatitis B immune globulin should be administered within 24 hours, and the hepatitis B vaccine should be offered to confer active immunity.
- There is no prophylaxis for exposure to hepatitis C.
- If the source patient is HIV positive, the HCW should be placed on a two- or three-drug regimen depending on the risk of HIV transmission. Prophylaxis should begin within 24 hours of exposure and be continued for 4 weeks if tolerated.

Risk Factors

- Use of large, hollow-bore needles without engineered protection
- Use of straight suture needles for securing vascular access devices
- Two-handed recapping of needles (13% of exposures in one study)
- Long work hours and fatigue have been reported to increase the risk of needle stick injuries in medical personnel.

Prevention

- Always use standard precautions.
- Do not recap needles if possible. If recapping is necessary, a one-handed scoop technique should be used.
- Needleless or protected needle devices should be used when available. Use blunt-tipped needles when possible.
- Sharp objects such as needles or scalpels with engineered safety systems should be used when practical.
- Needles should not be held while tying sutures.
- Barrier precautions (gloves, masks, eye shields) should be used whenever risk of exposure is present. Consider using a double-glove technique.

Special Considerations

A copy of the health care institution's occupational exposure protocol is maintained in the policy and procedures manual. A copy of this document should be available at all clinical locations. There is no need to modify the patient care responsibilities of an individual who has been exposed to hepatitis B or C or HIV, although the exposed individual should be counseled regarding infection control. Exposed health care workers should not donate blood, plasma, or tissue during the postexposure follow-up period. A health care worker who develops hepatitis B or C should modify his or her patient care responsibilities as recommended by the Centers for Disease Control and Prevention.

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Operating Room Fire

Definition

An infrequent but catastrophic event that can involve the airway, head and neck, or any other portion of the surgical field.

Presentation

- A puff of smoke and possibly a flash of light in an endotracheal tube
- Appearance of smoke on the surgical field or from under the drapes
- A “popping” sound may be heard.
- Flames may not be visible, especially if alcohol (from prep solutions) is the fuel.

Pathophysiology

Three elements must be present: fuel (e.g., alcohol or plastic), oxidizer (O_2 or N_2O), and an ignition source (electrocautery or laser). Operating room fires can produce significant amounts of

toxic smoke, but not enough heat to activate overhead sprinkler systems. Burning plastic (e.g., surgical drapes) can produce CO, hydrogen chloride (HCl), and cyanide.

Immediate Management

- Specific actions depend upon the location of the fire and source of fuel and oxidizer.
- **RACE: Rescue, Alert, Contain, Extinguish.**
- Rescue patients or staff in immediate danger.
- Activate the fire alarm.
- Contain the fire by removing the oxidizer from the fuel. Disconnect anesthesia circuit from patient for airway fire, discontinue O₂.
- Extinguish flames if it is safe to do so.
- Evacuate the room if necessary.
- **PASS: Pull** the pin to activate the extinguisher, **Aim** at the base of the fire, **Squeeze** the trigger, and **Sweep** the extinguisher back and forth across the fire.

299

DIFFERENTIAL DIAGNOSIS

There is no differential diagnosis. It is important to determine the ignition source, fuel, oxidizer, and location of the fire immediately.

Subsequent Management

- Subsequent management depends on the location of the fire.
- Determine whether evacuation of the operating room is necessary.
- Abort the surgical procedure as soon as it is safe to do so.

Risk Factors

- Use of laser surgical devices
- Use of electrocautery after application of alcohol-based prep solutions
- Use of O₂ enriched gas adjacent to electrocautery or laser
- Bringing hot items (i.e., halogen lamps or camera light sources) into contact with other flammable items

Prevention

- Minimize FiO₂ whenever surgery will take place near the airway.
- Be certain that flammable prep solutions have been removed from the patient or allowed to dry before using electrocautery.

- A response plan should be formulated before the patient is brought into the operating room. Know the location of fire extinguishers and fire blankets.

Special Considerations

There are three types of fire extinguishers: A (paper, cloth, plastics), B (liquids or grease), and C (electrical). Be sure to use the right extinguisher for the fire in progress. Most extinguishers in the OR are type ABC (can be used on all fires).

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Chapter 11

Surgical Emergencies

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Bleeding after Carotid Endarterectomy	302
Bleeding after Thyroid Surgery	304
Facial Trauma	307
Laparotomy in the Critically Ill Patient	309
Massive Hemorrhage	313
Neck Injury	316
Ruptured Abdominal Aortic Aneurysm	318
Ruptured Ectopic Pregnancy	322
Upper Gastrointestinal Bleeding	324

Bleeding after Carotid Endarterectomy

Definition

Bleeding from the carotid artery, internal jugular vein, smaller vessels, or raw tissue after carotid endarterectomy (CEA). Postoperative hemorrhage is rare (occurs in <1%–4% of patients).

Presentation

- In the awake patient, anxiety may be the first symptom of early airway compromise. Neurologic symptoms may rarely occur as a result of compression or disruption of the internal carotid artery.
- Tachycardia and tachypnea may occur, possibly due to anxiety. Decreased oxygen saturation is a late occurrence. Hypotension due to exsanguination from disruption of the suture line occurs rarely.
- New onset of dysphonia and stridor are diagnostic of airway compromise.
- An expanding hematoma may develop, which may or may not be pulsatile.

Anatomy and Pathophysiology

- Sites of operative hemorrhage after CEA include both venous and arterial bleeding at sites remote from the carotid artery itself. Diffuse microvascular bleeding may be caused by residual heparin effect or effects of antiplatelet drugs. The suture line of the endarterectomy site is another potential source of bleeding, usually presenting as diffuse oozing from the wound. In those instances in which vein-patch angioplasty is employed, the patch may rupture. Arterial bleeding may result in exsanguinating hemorrhage.
- An acute neck hematoma exerts pressure on the larynx and may cause life-threatening airway compromise.

Immediate Management of Acute Respiratory Distress

- Increase FiO_2 to 100%.
- Elevate the head of the bed.
- **Immediate re-exploration is mandatory.** Even a hematoma that initially appears benign can rapidly progress and compromise the airway. The surgical team should be called urgently to the bedside. Transport the patient to the operating room if the clinical situation permits.
- If the patient is in acute respiratory distress, it may be necessary to evacuate the hematoma first in order to intubate

Immediate Management (continued)

the trachea. Open the skin incision to reduce pressure on the trachea.

- If endotracheal intubation is impossible and airway obstruction is imminent, establishing a surgical airway may be necessary.

DIFFERENTIAL DIAGNOSIS

- Anaphylaxis
- Laryngeal edema
- Acute pulmonary edema
- Tension pneumothorax
- Acute coronary syndrome
- Pulmonary embolus

Diagnostic Studies

- This is primarily a clinical diagnosis that is made by clinical course and physical examination. Maintain a high index of suspicion in patients who have undergone this procedure.
- Because the patient can deteriorate rapidly, diagnostic studies (i.e., imaging) are not usually feasible. If the bleeding is slow and there is no acute airway compromise, carotid duplex ultrasound and diagnostic cervical ultrasound may be used to establish the diagnosis.

Subsequent Management

- After a definitive airway is established, transport the patient to the operating room (OR) for exploration of the operative site.
- Be prepared for a major vascular operation, including carotid isolation and repair.

Risk Factors

- Surgical technique
- Perioperative hypertension
- Perioperative use of aspirin or other antiplatelet agents
- Failure to reverse heparin with protamine sulfate
- Postoperative anticoagulation with heparin or warfarin
- The use of a small vein may increase the risk of rupture after a vein patch procedure.

Prevention

- Meticulous hemostasis at the time of the original operation
- Systolic blood pressure should be <140 mm Hg in normotensive patients and 160 mm Hg in chronically hypertensive patients.
- Consider administering protamine (1–1.5 mg per 100 units of heparin, not to exceed 50 mg) to reverse heparin used during the procedure. This has been demonstrated to reduce postoperative bleeding complications without influencing the incidence of postoperative stroke.
- The risk of vein patch rupture can be minimized by using greater saphenous vein harvested from the thigh.

Special Considerations

- The incidence of reoperation for hematoma drainage is <1%, but this complication is potentially life-threatening because of associated airway compromise or, rarely, exsanguination from arterial suture line disruption.

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Bleeding after Thyroid Surgery

Definition

Bleeding from the thyroid surface, dissected muscles, blood vessels in the vicinity of the thyroid gland, or the thyroid vessels themselves after thyroid surgery.

Presentation

- In the awake patient, anxiety may be the first symptom of early airway compromise.
- Tachycardia and tachypnea due to anxiety or airway compromise may occur. Decreased oxygen saturation is a late occurrence.
- New onset of dysphonia and stridor indicate airway compromise.

- A new neck mass in proximity to the operative site, which may or may not be associated with bleeding from the incision, represents a hematoma until proved otherwise.

Pathophysiology

- Sources of postoperative hemorrhage after thyroid surgery include the raw surfaces of residual thyroid tissue, venous bleeding, and arterial bleeding. The bleeding may originate from vessels deep or superficial to the cervical strap musculature. Large goiters may also have associated enlarged mediastinal vessels, which include the internal mammary artery, the thyroidea ima artery, and the innominate vein.
- When an acute hematoma occurs in the neck, the resulting pressure results in compression of the larynx, which can lead to life-threatening airway compromise.
- Up to 3% of patients with thyroid diseases undergoing thyroid surgery have various acquired abnormalities of coagulation. The most common abnormality resembles von Willebrand disease. Patients who have hypothyroidism are the most likely to have an abnormal bleeding tendency, but coagulopathy may also occur in patients with thyroid malignancies.

DIFFERENTIAL DIAGNOSIS

- Anaphylaxis
- Laryngeal edema
- Acute pulmonary edema
- Tension pneumothorax
- Acute coronary syndrome
- Pulmonary embolus

Immediate Management

- Increase FiO_2 to 100%.
- **Immediate re-exploration is mandatory.** Even a hematoma that initially appears benign can rapidly progress and compromise the airway. The surgical team should be called urgently to the bedside. Transport the patient to the OR if the clinical situation permits.
- If endotracheal intubation is not possible and the patient is in acute distress, a laryngeal mask airway (LMA) may be attempted as a bridge to definitive airway management. Note: If the obstruction is infraglottic, an emergency surgical airway may be required.

Immediate Management (continued)

- After the hematoma is evacuated and laryngeal anatomy is restored, endotracheal intubation is usually possible. A surgical airway may rarely be necessary if endotracheal intubation is not possible after hematoma evacuation.

Diagnostic Studies

- Physical examination and a high index of suspicion.
- The urgency of the situation precludes imaging or other diagnostic studies.

Subsequent Management

After a definitive airway is established, the surgical site should be explored and the source of hemorrhage controlled.

Risk Factors

- Surgical technique is the most common cause, particularly poor hemostasis.
- Extensive surgery, which can include resection of large goiters
- Underlying bleeding diathesis or the use of anticoagulants (e.g., heparin, warfarin, clopidogrel)
- Male gender
- Advanced age

Prevention

Meticulous hemostasis at the time of surgery. Preoperative coagulation screening to identify patients at increased risk of bleeding. Early detection and treatment of reversible coagulopathy. A drain can be placed in the resection bed to detect early signs of hemorrhage, especially if the patient is obese and body habitus makes it difficult to identify a cervical hematoma.

Special Considerations

- Although bleeding and wound hematomas have an occurrence rate of <1%, these complications may be life-threatening due to airway compromise.

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Facial Trauma

Definition

Maxillofacial injuries may be dramatic in appearance but are rarely life-threatening, unless airway, breathing, or circulation are compromised as a result of the injury.

Presentation

- Compromise of airway and breathing:
 - Signs of impending respiratory obstruction include stridor, cyanosis, drooling, or ineffective gag reflex. Note: Airway obstruction may result in early death in the setting of multiple mandibular fractures or the combination of nasal, maxillary, and mandibular fractures.
 - Aspiration of teeth, blood, vomitus, or foreign bodies may cause airway obstruction or pneumonitis.
 - Patients may present with significant airway obstruction and impending respiratory failure despite normal pulse oximetry values.
- Extensive hemorrhage:
 - Epistaxis
 - Scalp lacerations
 - Tongue lacerations
 - LeFort fractures

Anatomy and Pathophysiology

Blunt trauma may cause pan-facial fractures, significant soft tissue injury, and multiple remote sites of injury. Penetrating trauma ranges from simple lacerations to high-velocity missile injuries that may also involve the brain or neck.

Associated Injuries

- Airway obstruction
- Brain injury
- Cervical spine injury
- Hemorrhage from other sites

Immediate Management

- Secure the airway: An emergency surgical airway may be necessary if orotracheal intubation is not possible. (Note: A cricothyroidotomy takes significantly less time than a tracheostomy and should be the surgical airway of choice if respiratory failure is imminent.) Nasotracheal intubation is contraindicated. If trained personnel are available and a cricothyroidotomy is not feasible, retromolar intubation or submental intubation may be considered.
- Maintain cervical spine stabilization during airway management, with inline immobilization and minimal extension.
- Establish large-bore peripheral intravenous access and begin fluid resuscitation.
- Ask the surgical team to obtain local control of hemorrhage.
- Assess the patient for concomitant injury.

308

Diagnostic Studies

A computed tomography (CT) scan is the standard of care. Three-dimensional reconstruction of axial CT sections should be requested if available. Panorex films are a useful adjunct for defining mandible fractures by providing a two-dimensional or linear image typically sufficient for diagnosis of fractures and operative planning.

Subsequent Management

- Management of associated life-threatening injuries is undertaken first.
- Early tracheostomy should be considered in selected patients as follows:
 - Pan-facial fractures
 - Profuse nasal bleeding
 - Severe soft tissue edema in the proximity of the airway
 - Patients with altered mental status
 - Severe facial burns
 - High spinal cord injuries
 - Difficult airway characteristics
 - Need for prolonged intubation
- Assume that the cervical spine is unstable. A cervical collar should remain in place until definitive clearance by a combination of physical examination and radiographic examination.

- Definitive management of facial injuries, particularly facial fractures, is usually delayed until life-threatening injuries are managed and the patient is stable.

Prevention

Maxillofacial injuries themselves are seldom life-threatening. Associated injuries, however, are serious and must be managed first in order to prevent loss of life.

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Laparotomy in the Critically Ill Patient

Definition

Emergency laparotomy in a critically ill patient for one or more life-threatening conditions, including infection, hemorrhage, ischemia, and abdominal compartment syndrome.

Presentation

- Signs and symptoms vary with etiology and indication for laparotomy.
- Shock (hemorrhagic, septic, or traumatic) is usually present.
- End-organ perfusion may be impaired; signs include altered mental status, oliguria, tachypnea, hyperlactacidemia, and increased base deficit.
- Significant tissue hypoperfusion can occur in spite of a normal arterial blood pressure. The presence of hypotension implies severe physiologic decompensation.

Pathophysiology

- *Shock* is defined as the inadequate delivery of oxygen and nutrients to tissues as a result of hypoperfusion. Shock is characterized in various ways based on etiology. A clinically useful scheme is as follows:

- Hypovolemic/hemorrhagic (acute hemorrhage of any etiology, peritonitis, pancreatitis, bowel obstruction)
- Vasodilatory/distributive (sepsis, adrenal insufficiency, high spinal cord injury, liver failure, anaphylaxis)
- Cardiogenic (myocardial infarction, tamponade, arrhythmias)
- Obstructive (pulmonary embolus, pneumothorax)
- Traumatic (a combination of hemorrhage, ischemia, reperfusion, activation of proinflammatory cascades)
- Persistent hypothermia and progressive metabolic acidosis in the setting of massive transfusion are associated with life-threatening coagulopathy.

DIFFERENTIAL DIAGNOSIS

- Cardiovascular collapse from an extraabdominal source of sepsis
- Hemorrhage from extraabdominal trauma (pelvic fractures, long-bone fractures, thoracic injuries, open extremity wounds, scalp lacerations)
- Massive myocardial infarction with acute cardiac failure and cardiovascular collapse
- Massive pulmonary embolus with cardiovascular collapse

Immediate Management

- Intubate the trachea and initiate mechanical ventilation. Consider etomidate (0.3 mg/kg IV) for induction if the patient is hemodynamically unstable. Sympathomimetic agents (e.g., ketamine) may cause profound hypotension in critically ill patients who have high circulating levels of endogenous catecholamines.
- Increase FiO_2 to maintain adequate oxygenation.
- Be prepared to support the blood pressure. Hypotension is exacerbated by the transition from spontaneous ventilation to positive pressure mechanical ventilation.
- Establish large-bore peripheral or central venous access.
- Begin aggressive resuscitation with IV fluids. Transfuse with packed red blood cells (PRBCs) if indicated (i.e., for hemorrhagic shock).
- Promptly identify and correct coagulopathy, thrombocytopenia, and platelet dysfunction.
- Manage hypothermia.
- Support blood pressure with vasopressors such as norepinephrine if indicated (septic shock).
- Consider epinephrine infusion starting at 0.03–0.05 mcg/kg/min.
- Begin broad-spectrum empiric or culture-directed antibiotic therapy (septic shock).

Diagnostic Studies

- Abdominal and pelvic CT scan is the standard for diagnosis of intra-abdominal catastrophes.
- If the patient is too unstable for CT scan, emergency surgery may be indicated based on a clinical diagnosis.
- In the unstable trauma patient, focused abdominal sonography for trauma (FAST) and diagnostic peritoneal lavage can be quickly performed in the trauma bay to confirm intra-abdominal hemorrhage.

Subsequent Management

- Maintain close communication with the surgical team throughout the perioperative period.
- Hemodynamic instability may be caused by surgical manipulation and the patient's underlying pathophysiology. A narcotic-based technique is associated with less vasodilation and negative inotropy and may be appropriate for patients who are hemodynamically unstable or who have underlying myocardial dysfunction. Pay close attention to the patient's volume status, body temperature (fluid warmer, forced hot air blanket, elevated room temperature are most effective), and prevention of positioning injuries.
- Patients undergoing emergency laparotomy are more likely to have hemodynamic instability in the postoperative period. Most patients remain intubated and mechanically ventilated after surgery until the patient has a stable pulmonary and hemodynamic status. Depending on the degree of physiologic impairment, many patients require prolonged mechanical ventilation and ongoing resuscitation in the ICU.

Risk Factors

- The lethal triad: hypothermia, acidosis, coagulopathy
- Advanced age
- Chronic illness; comorbidities

Prevention

Early diagnosis of an impending intra-abdominal catastrophe before the patient goes into shock may prevent significant hypoperfusion and associated end-organ dysfunction, and may increase the likelihood of a good outcome.

Special Considerations

- Intraoperative management of the critically ill patient is centered on maintaining oxygenation and perfusion. The choice of a

specific technique is guided by the patient's volume status and medical condition.

- Abdominal compartment syndrome:
 - Abdominal hypertension with associated end-organ dysfunction (most commonly oliguria and elevated peak airway pressures)
 - Bladder pressure is used to estimate intra-abdominal pressure.
 - Etiologies include blunt and penetrating abdominal trauma, pelvic fractures, severe burns, massive resuscitation, and ischemia-reperfusion of the abdominal viscera.
 - Treatment is abdominal decompression, typically via decompressive laparotomy, leaving the abdomen open.
 - Mortality rates of 60%–70% reflect delayed diagnosis and underlying pathophysiology.
 - Early decompression is essential to prevent irreversible end-organ ischemia and cardiovascular collapse.
- Damage control surgery:
 - Damage control is the preferred strategy for trauma patients with abdominal injuries complicated by hypothermia, coagulopathy, and acidosis. This strategy can also be used for critically ill nontrauma patients.
 - Assess physiologic reserve and severity of the physiologic insult.
 - Limited initial operation for the immediate control of hemorrhage and contamination; the abdomen is left open with a temporary abdominal closure in place.
 - Ongoing resuscitation and correction of physiologic derangements occurs in the ICU.
 - Subsequent operation for correction of anatomic abnormalities occurs in the stabilized patient, and early abdominal closure results in improved postoperative outcomes.

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Massive Hemorrhage

Definition

- Major categories of massive hemorrhage include traumatic, gastrointestinal, and obstetric.
- Classes of hemorrhagic shock:
 - Class I: blood loss up to 750 cc (up to 15% of blood volume)
 - Heart rate <100
 - Blood pressure normal
 - Pulse pressure normal or increased
 - Class II: blood loss 750–1500 cc (15–30% of blood volume)
 - Heart rate >100
 - Blood pressure normal
 - Pulse pressure decreased
 - Class III: blood loss 1500–2000 cc (30–40% of blood volume)
 - Heart rate >120
 - Blood pressure decreased
 - Pulse pressure decreased
 - Class IV: blood loss >2000 cc (>40% of blood volume)
 - Heart rate >140
 - Blood pressure decreased
 - Pulse pressure decreased

Presentation

- Symptoms depend on the etiology of the hemorrhage. Gastrointestinal blood loss is typically painless; blood loss associated with trauma is associated with pain due to the injury.
- Perturbation of vital signs depends on the degree of blood loss.
- Severe shock is associated with cool, moist, pallid, or cyanotic skin. Mental status changes progress from anxiety to confusion to lethargy as the degree of shock progresses. Tachypnea occurs as the spontaneous minute ventilation increases in response to increasing metabolic demands. Oliguria is caused by renal hypoperfusion.
- The source of hemorrhage may or may not be visible on external examination.

Pathophysiology

Hemorrhagic shock is a complex spectrum of events:

- Acute massive blood loss resulting in circulatory collapse
- Ischemia-reperfusion injury
- Inflammatory and anti-inflammatory responses
- Multiple organ dysfunction

Immediate Management

- Intubate the trachea and initiate mechanical ventilation.
- Establish large-bore peripheral and central IV access.
- Transfuse with packed red blood cells and factors as indicated. Consider activating the massive transfusion protocol.
- Begin resuscitation with crystalloid solutions (e.g., lactated Ringer's solution or normal saline solution)
- If laboratory studies are not feasible, resuscitate the patient with plasma, PRBCs, and platelets in a 1:1:1 ratio and minimal amounts of crystalloid solutions. Administer cryoprecipitate in the setting of continued microvascular bleeding.
- If time permits, identify and correct specific deficits with serial prothrombin time (PT), partial thromboplastin time (PTT), international normalized ratio (INR), fibrinogen, and platelet count. Elevated INR predicts the requirement for massive transfusion therapy.

DIFFERENTIAL DIAGNOSIS

Includes other causes of acute circulatory collapse:

- Hypovolemic shock of nonhemorrhagic etiology (bowel obstruction, pancreatitis)
- Vasodilatory/distributive shock
- Obstructive shock
- Cardiogenic shock

Diagnostic Studies

- Gastrointestinal source: Endoscopy, interventional radiology, nuclear medicine.
- Traumatic source: CT scan, FAST, diagnostic peritoneal lavage, immediate operative intervention in hemodynamically unstable patients.
- Obstetric source: Usually apparent on physical examination or ultrasound examination.

Subsequent Management

- Prompt surgical control of the bleeding.
- Continue resuscitation throughout the perioperative period.

- The goals of resuscitation are optimization of preload, cardiac performance, blood pressure, oxygen delivery and end-organ perfusion. No single parameter is universally applicable to every patient. Therefore, multiple endpoints should be optimized:
 - Clinical endpoints (heart rate, respiratory rate, blood pressure, urine output, level of consciousness, pulse pressure)
 - Cardiac output measurement
 - Metabolic parameters (lactate, base deficit)
 - Regional perfusion (gastric tonometry, sublingual capnography, near-infrared spectroscopy)
- Massive transfusion protocols include component therapy, which replaces loss of blood volume, restores tissue perfusion, and corrects coagulopathy.
- Fresh-frozen plasma produces less inflammation than do crystalloid or colloid solutions.

Risk Factors

- GI source: Advanced age, comorbidities.
- Traumatic source: Young age (injury is the leading cause of death for persons younger than 44 years of age in the US) and lifestyle issues.
- Obstetric source: Postpartum hemorrhage is most commonly caused by uterine atony.

315

Prevention

Shock, hypoperfusion, and organ dysfunction are prevented by early control of the bleeding and appropriate resuscitation, regardless of the etiology of the hemorrhage.

Special Considerations

- Massive transfusion is usually defined as the complete replacement of the patient's entire blood volume—10 units PRBC—in a 24-hour period.
- Role of recombinant-activated factor VII (rFVIIa):
 - Approved in the United States only for bleeding associated with hemophilia.
 - It is frequently used off-label, including for the reversal of the coagulopathy of trauma.
 - Generates a thrombin peak, which in turn causes formation of a fibrin plug.
 - May not be efficient in patients with acidosis (consider biochemical correction of acidosis prior to administration).
 - Hypothermia has little effect on efficacy.

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Neck Injury

316

Definition

The neck contains a high density of vital structures located in a small and unprotected area. This may result in multisystem injury as a consequence of a single traumatic event.

Presentation

- “Hard” signs warrant urgent surgical intervention:
 - Active bleeding
 - Expanding or pulsatile hematoma
 - Subcutaneous emphysema or air bubbling from wound
 - Airway compromise
 - Hematemesis
- “Soft” signs warrant a more selective or expectant approach:
 - Dysphagia
 - Voice change
 - Hemoptysis
 - Wide mediastinum

Anatomy and Pathophysiology

- Structures located in the neck:
 - Sternocleidomastoid muscles
 - Carotid artery, internal jugular vein, and vertebral artery
 - Pharyngeal-esophageal junction
 - Larynx and proximal trachea

- Thyroid and parathyroid glands
- Thoracic duct—enters the jugulosubclavian system in the left neck
- Cervical vertebra and spinal cord
- Long cervical musculature (posterior)
- Anterior triangles of the neck: the area between the sternocleidomastoid muscles
- Zones of the neck:
 - Zone I: the area bounded by the cricoid cartilage superiorly, the thoracic inlet inferiorly and the sternocleidomastoid laterally
 - Zone II: the area between the cricoid cartilage and the angle of the mandible
 - Zone III: the area bounded by the angle of the mandible inferiorly and the base of the skull superiorly

Associated Injuries

- Spinal cord injury
- Brain injury
- Facial trauma

Immediate Management

- **Secure the airway if it is compromised.** Orotracheal intubation is preferred; a surgical airway should be created if orotracheal intubation is not possible.
- Maintain cervical spine stabilization during airway management.
- Establish large-bore IV access. If central venous access is required, consider cannulating the femoral vein.
- Resuscitate with crystalloid and PRBC if indicated.
- Control active hemorrhage with local application of pressure. Balloon catheter tamponade may be required if manual pressure does not minimize hemorrhage while the patient is transported to the operating room.

Diagnostic Studies

- Selected radiographic and endoscopic studies (see the following).
- Immediate surgery in the setting of active hemorrhage.

Subsequent Management

- Formal neck exploration is indicated in the presence of “hard” signs that indicate major vascular or aerodigestive tract injury.
- Penetrating wounds should only be examined in the operating room as part of a formal neck exploration, and not in the emergency department.

- In the absence of “hard” signs, a selective approach for management may be considered. This includes a thorough physical examination and specific diagnostic studies to identify vascular and aerodigestive tract injuries:
 - Esophagoscopy
 - Radiologic examination of the esophagus
 - Laryngoscopy/tracheoscopy/flexible nasoendoscopy
 - Arteriography (conventional and CTA)
 - Doppler ultrasonography
 - Computed tomography scan
- Surgical treatment of identified injuries
- Endovascular treatment of internal carotid injuries using stents and coils is gaining in acceptance. These techniques can often be used to treat the majority of vertebral artery injuries.

Special Considerations

Penetrating neck injuries are frequently life threatening. Immediate protection of the airway and rapid control of exsanguinating hemorrhage may be necessary.

318

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Ruptured Abdominal Aortic Aneurysm

Definition

Rupture is a potentially lethal complication of abdominal aortic aneurysm (AAA).

Presentation

- Most patients who present with a ruptured AAA are unaware that they have aneurysmal disease. Patients may present with a symptomatic aneurysm (pain without evidence of rupture), a contained leak, or free rupture.
- The classic clinical presentation of AAA rupture includes abdominal pain, hemodynamic instability, and a pulsatile abdominal mass. The abdominal pain is usually acute and

unremitting, radiating to the back. If the rupture is in the retroperitoneum adjacent to the ureter, pain may be referred to the ipsilateral testicle or groin.

- The patient may experience light-headedness or collapse because of acute hypovolemia.
- If the aneurysm perforates into the duodenum or colon, massive gastrointestinal hemorrhage may occur.
- If rupture occurs directly into the inferior vena cava, high-output congestive heart failure results.

Pathophysiology

Once an aneurysm develops, regardless of the etiology, enlargement is governed by the Law of Laplace, $T = PR$. T is tangential stress (which disrupts the wall of a sphere), P is the transmural pressure, and R is the radius. Large aneurysms are therefore more likely to rupture than small ones.

Immediate Management

- Intubate the trachea if the patient is in severe shock and is unable to protect the airway.
- If the clinical situation permits, transport the patient to the operating room and induce anesthesia after the patient is prepped and draped, because of the possibility of severe hypotension. Establish large-bore peripheral and central venous access.
- Initiate aggressive resuscitation with IV fluids and PRBC.
- Patients who present with the classic triad—pain, hemodynamic instability, and a pulsatile abdominal mass—must be transferred immediately to the operating room while being resuscitated. Any delay in control of the hemorrhage may be life threatening. Patients may undergo open or endovascular treatment.

319

DIFFERENTIAL DIAGNOSIS

- Acute infectious or inflammatory abdominal process (hollow viscus rupture, ischemic or infarcted bowel, acute cholecystitis, acute pancreatitis) causing septic shock or exaggerated systemic inflammatory response
- Hemorrhage from another intra-abdominal source, including ruptured visceral artery aneurysm, solid organ rupture (liver, spleen, kidney), hepatobiliary tumor hemorrhage
- Massive gastrointestinal hemorrhage
- Aortic dissection or aortic occlusion
- Massive myocardial infarction with resultant acute cardiac failure, cardiogenic shock, and hemodynamic collapse
- Massive pulmonary embolus with resultant obstructive shock and hemodynamic collapse

Diagnostic Studies

- Hemodynamically stable patients should undergo CT of the abdomen and pelvis with intravenous contrast.
- Duplex ultrasound (which may be performed at the bedside) can rapidly determine the presence of an AAA but may not image all portions of the aortic wall and therefore cannot identify ruptured versus nonruptured AAA.

Subsequent Management

- All patients with ruptured AAA require urgent surgery or endovascular treatment.
- Hemodynamically unstable patients must undergo immediate surgery or endovascular treatment. The patient is resuscitated during the surgical procedure because operative intervention cannot be delayed.
- Assess and treat coagulopathy, thrombocytopenia, and platelet dysfunction. Baseline laboratory studies include CBC, PT, PTT, comprehensive chemistry panel, and cardiac enzymes. Coagulopathy is treated with fresh-frozen plasma and platelets, as indicated by laboratory parameters.
- In hemodynamically stable patients, timing of the repair depends upon CT scan results. Emergency surgical management is necessary for a contained peritoneal rupture. In symptomatic patients who do not demonstrate CT evidence of rupture, repair can be postponed for up to 24 hours while the patient's medical condition is optimized.
- Endovascular repair (EVAR) has been demonstrated to decrease 30-day mortality and increase long-term survival for treatment of ruptured AAAs compared to open surgical repair. Although EVAR is associated with a decreased morbidity and mortality as compared to conventional surgery, there is an increased risk of reintervention. Depending on surgeon expertise, EVAR can be performed using local anesthesia for a percutaneous approach.
- Monitor the patient for abdominal compartment syndrome after EVAR.

Risk Factors

- The most important risk factor for rupture is the maximum diameter of the aneurysm. Aneurysms 4.0–5.4 cm in diameter have a yearly risk of rupture of 0.5–1%. Aneurysms 6–7 cm have a 6.6% risk of rupture per year.
- Hypertension and hyperlipidemia
- Chronic obstructive pulmonary disease
- Smoking

Risk Factors (continued)

- Female gender
- Eccentric saccular aneurysms
- Rate of expansion of the AAA as an independent risk factor has been implicated but not proved.

Prevention

- Avoid abrupt episodes of hypertension until the aneurysm has been secured.
- The goal of elective repair of AAAs is to avoid rupture. The mortality of elective repair is 6% compared with >48% for repair of ruptured AAA. Endovascular repair confers a lower perioperative mortality rate (16%–31%) compared to open repair for ruptured AAA.
- The overall mortality rate for ruptured AAA is 90%, since 60% of patients die prior to reaching the operating room.
- Elective operative repair is recommended for AAAs 5.5 cm or greater in males and 4.5–5.0 cm in females and patients with greater than average rupture risk.
- Rate of growth is an important indicator for surgical intervention, 5–7 mm per 6 months or >1 cm per year.

Special Considerations

- More than 90% of aneurysms are associated with atherosclerosis, but 75% of patients with aneurysmal disease do not have occlusive vascular disease. Multiple factors contribute to the destruction of the media of the aortic wall, leading to aneurysm formation. Alterations in the connective tissue of the aortic wall, proteolytic enzymes, and inflammatory changes have been implicated.
- Abdominal aortic aneurysms are less frequently caused by infection, arteritis, cystic medial necrosis, trauma, inherited connective tissue disorders, and pseudoaneurysm formation. Abdominal aortic aneurysms in young adults and children occur in the setting of tuberous sclerosis, Behcet disease, Marfan syndrome, Ehlers-Danlos syndrome, and infection associated with umbilical artery catheters.

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Ruptured Ectopic Pregnancy

Definition

- Implantation of a fertilized ovum outside of the endometrial cavity. In ruptured tubal ectopic pregnancy, trophoblastic proliferation extends through the tube. Hemorrhage occurs when the pregnancy extends into the large blood vessels in the broad ligament.

Presentation

- Classic symptoms: Abdominal or pelvic pain and vaginal bleeding with an associated positive pregnancy test. Pain radiating to the shoulder, syncope, and shock are caused by hemoperitoneum secondary to ruptured ectopic pregnancy and occur in 20% of patients.
- Hemorrhagic shock dependent on the degree of blood loss (see Massive Hemorrhage).
- Abdominal tenderness in 90% of patients. Peritonitis in 70% (typically rebound tenderness). Cervical motion tenderness is present in approximately 30% of patients, whereas a tender adnexal mass is evident in up to 50%.

Pathophysiology

In tubal implantations (the most common type of ectopic gestation), the proliferating trophoblast first invades the luminal mucosa, then the muscularis and lamina propria, and finally the serosa. Invasion into the large blood vessels in the broad ligament results in hemorrhage that distorts the tube and causes pain. Although some ectopic pregnancies are clinically silent and end with spontaneous tubal abortion, rapid, life-threatening hemorrhage can occur.

Immediate Management

- Establish large-bore peripheral IVs and/or central venous access.
- Begin aggressive fluid resuscitation. Class I and Class II hemorrhagic shock can usually be treated with crystalloid infusions. Transfuse PRBCs in patients with Class III and Class IV shock (see Massive Hemorrhage).

DIFFERENTIAL DIAGNOSIS

- Early diagnosis of hemorrhage is critical. The patient will not develop hypotension until 30% of the circulating blood volume is lost. Mild tachycardia may be the first sign of significant blood loss.
- Infectious or inflammatory intra-abdominal and pelvic processes involving the gastrointestinal tract (perforated viscus, peptic ulcer disease, intestinal ischemia, appendicitis, colitis, cholecystitis, diverticulitis, pancreatitis), the urinary tract (urosepsis of any etiology, including pyelonephritis, cystitis, obstructive nephroureterolithiasis), and the reproductive tract (pelvic inflammatory disease, salpingitis, endometritis).
- Hemorrhagic intra-abdominal and pelvic processes, including ruptured solid organ (liver, spleen, kidney), ruptured aortic or visceral aneurysm, ruptured hemorrhagic ovarian cyst, and uterine rupture (may occur with traumatic injury).

Diagnostic Studies

- In patients who have hemorrhagic shock without a previous diagnosis of ectopic pregnancy, a single beta-hCG measurement and ultrasound guides definitive management.
- Routine tests to establish ectopic pregnancy include serial measurements of beta-hCG, ultrasonography, uterine (endometrial) sampling, and occasionally progesterone levels.

Subsequent Management

- Resuscitation is guided by CBC, PT, PTT, and INR. For massive hemorrhage, check baseline fibrinogen level and obtain periodic fibrinogen levels. Infusion of fresh-frozen plasma, cryoprecipitate, and platelets is guided by laboratory parameters and surgical bleeding.
- Either laparotomy or laparoscopy with salpingectomy is performed for ruptured ectopic pregnancy.
- If the patient is in severe hemorrhagic shock and rapid entry into the peritoneal cavity for source control is needed, emergency laparotomy is usually the best choice.

Risk Factors

- High risk: Tubal surgery, tubal ligation, previous ectopic pregnancy, in utero exposure to diethylstilbestrol (altered fallopian tube development), presence of an intrauterine device, tubal pathology, assisted reproduction.
- Moderate risk: Infertility, previous genital infections, multiple sexual partners.
- Low risk: Previous pelvic infection, cigarette smoking, vaginal douching, young age at first intercourse.

Prevention

Risk stratification and a high index of suspicion can be used to diagnose an ectopic pregnancy before it ruptures and hemorrhages.

Special Considerations

- The most common site of ectopic pregnancy is the fallopian tube (approximately 98% of all ectopic gestations). Abdominal, ovarian, and cervical ectopic pregnancies comprise the remaining 2%. *Heterotopic pregnancy* is the simultaneous occurrence of intrauterine and extrauterine gestation.
- Serial beta-human chorionic gonadotropin (beta-hCG) levels are used in diagnostic algorithms. Progesterone levels are lower in ectopic pregnancies than in intrauterine gestations, but there is no established cutoff that can distinguish between the two.
- Although multiple-dose systemic methotrexate is the first-line medical treatment of ectopic pregnancy, operative management is always the treatment of choice for a ruptured ectopic pregnancy.

Further Reading

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Upper Gastrointestinal Bleeding

Definition

Upper gastrointestinal (UGI) hemorrhage originates proximal to the ligament of Treitz and accounts for almost 80% of major hemorrhage. Bleeding sites include the esophagus, stomach, duodenum, and, more rarely, the hepatobiliary tree, pancreas, and aortoenteric fistulae.

Presentation

- Hematemesis and/or melena. Abdominal pain is rare.
- Hemorrhagic shock. Classification is dependent on the degree of blood loss (see Massive Hemorrhage). Manifestations of shock, such as tachypnea, oliguria, and mental status changes may be present. In patients presenting with variceal hemorrhage, signs of portal hypertension and jaundice may be noted.

Anatomy and Pathophysiology

- Peptic ulcer disease is the most common etiology of UGI hemorrhage and accounts for >50% of cases. *Helicobacter pylori* and nonsteroidal anti-inflammatory drugs (NSAIDs) are implicated in most cases.
- Gastric and esophageal varices are the second most common etiology and account for 15% of cases of UGI hemorrhage. Cirrhosis causes portal hypertension, which in turn results in formation of varices.
- Less common etiologies include stress ulceration, esophagitis, Mallory-Weiss tear, Dieulafoy's lesion, arteriovenous malformations, and tumors. Unusual etiologies include hemobilia and hemosuccus pancreaticus; unlike most patients presenting with UGI hemorrhage, these patients may have abdominal pain. Aortoenteric fistula is another infrequent etiology of UGI hemorrhage.
- The most predictive factors of severity include history of malignancy or cirrhosis, fresh blood hematemesis, hypovolemic signs including hypotension, tachycardia, shock, and Hb <8 g/dL at initial presentation.

DIFFERENTIAL DIAGNOSIS

Lower gastrointestinal hemorrhage originates below the ligament of Treitz and is generally less life threatening than UGI hemorrhage; shock is less likely and transfusion requirements are typically lower. Eighty percent of all patients with gastrointestinal hemorrhage pass blood in some form from the rectum. Twenty percent of all cases of apparent lower gastrointestinal hemorrhage have an upper gastrointestinal source, including massive nasal or oropharyngeal hemorrhage resulting in swallowed blood.

Immediate Management

- Intubate the trachea and initiate mechanical ventilation if the patient has hematemesis or altered mental status, or if shock is imminent.
- Be prepared to suction copious blood from the airway.
- Establish large-bore peripheral and/or central venous access.
- Resuscitate aggressively with crystalloid IV fluids and PRBC. Class I and Class II hemorrhagic shock usually can be treated with crystalloid infusions. Packed red blood cells are necessary in Class III and Class IV shock.
- Draw baseline laboratories: CBC, PT, PTT, INR, and comprehensive metabolic panel, including liver function tests.

Immediate Management (continued)

- In cases of massive hemorrhage or known hepatic dysfunction, check fibrinogen level at baseline and periodically throughout the resuscitation.
- Correct coagulopathy, thrombocytopenia, platelet dysfunction (medication induced or pathologic) with blood products and ddAVP.

Diagnostic Studies

- After initial stabilization, determine the source of bleeding. Esophagogastroduodenoscopy is the modality of choice. Findings of bleeding peptic ulcers are useful in predicting the risk of rebleeding:
 - Active arterial bleeding
 - 90%–100% risk of rebleeding without endoscopic intervention
 - 15%–30% risk of rebleeding with endoscopic intervention
 - Visible nonbleeding vessel
 - 40%–50% risk of rebleeding without endoscopic intervention
 - 15%–30% risk of rebleeding with endoscopic intervention
- Adherent clot in ulcer base
 - 20%–30% risk of rebleeding without endoscopic intervention
 - 5% risk of rebleeding with endoscopic intervention
- Angiography: Bleeding rates must be at least 0.5 mL/min for adequate visualization.
- Nuclear medicine scans (tagged red blood cell scan): Bleeding rates must be at least 0.1 mL/min to be detected.

Subsequent Management

- Interventional radiology may be used to deliver intra-arterial vasopressin or embolize the lesion in poor surgical candidates.
- Restrictive transfusion strategies (threshold of Hb 7 g/dL) have been demonstrated to decrease complications, transfusion requirements and mortality compared to liberal transfusion (threshold of Hb of 9 g/dL).
- Surgical indications in patients with UGI hemorrhage attributable to peptic ulcer disease include two failed attempts of endoscopic hemostasis, rapid deterioration attributable to exsanguination, large visible vessels not amenable to endoscopic coagulation, and documented malignant ulcers.
- Medical management of variceal hemorrhage includes vasopressin (bolus 0.4 U with 0.4–1 U/min infusion) plus nitroglycerin (10–50 mcg/min) or octreotide (50 mcg bolus with 50 mcg/h infusion for 5 days).

- Endoscopic management of variceal hemorrhage includes sclerotherapy and variceal band ligation.
- Esophageal variceal bleeding may be treated with transjugular intrahepatic portosystemic shunt (TIPS) when medical and endoscopic therapy fail or are not feasible, as a bridge to hepatic transplantation. There is an associated risk of worsening hepatic encephalopathy.

Risk Factors for Mortality (6%–10%)

- Advanced age
- Renal insufficiency
- Hepatic failure
- Disseminated malignancy

Prevention

Treatment of *Helicobacter pylori* infection and limitation of NSAID use may help to prevent complications of peptic ulceration. Major indications for stress ulcer prophylaxis in the critically ill include respiratory failure and coagulopathy.

Special Considerations

- In general, 80% of cases of UGI hemorrhage stop spontaneously. Ninety percent hemostasis rates are achieved with endoscopic therapy. Surgical indications in patients with UGI hemorrhage attributable to peptic ulcer disease include two failed attempts of endoscopic hemostasis, rapid deterioration attributable to exsanguination, large visible vessels not amenable to endoscopic coagulation, and documented malignant ulcers. In patients with UGI hemorrhage and cirrhosis, prophylactic antibiotics have been demonstrated to reduce bacterial infections and increase survival. Norfloxacin 400 mg BID for 7 days has been recommended.

Further Reading

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Chapter 12

Postanesthesia Care Unit

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Altered Mental Status	330
Chest Pain	332
Hypoxia	335
Myocardial Ischemia	337
Oliguria/Acute Renal Failure	339
Postoperative Hypertension	341
Postoperative Hypotension	343
Postoperative Nausea and Vomiting	346
Prolonged Neurologic Impairment after Regional Anesthesia	348
Respiratory Depression or Failure	349
Severe Postoperative Pain	352
Stroke	354

Altered Mental Status

Definition

A clinical spectrum that includes inappropriate or unexpected behaviors, ranging from emergence delirium through delayed awakening.

Presentation

- Somnolence, disorientation, and mental sluggishness are common immediately after emergence.
- Emergence excitation may result in a highly agitated state that includes combativeness, confusion, and disorientation that is resistant to treatment.
- *Delayed awakening* occurs when a patient fails to respond to stimulation within 30 minutes following anesthesia with no obvious underlying cause.

Pathophysiology

Changes in mental status after surgery are generally not the result of organic brain disease. Pain may cause agitation, confusion, and aggressive behavior during emergence. Endotracheal tubes, drains, and catheters, as well as gastric and urinary distention cause discomfort and may lead to agitation or combativeness. Confusion and delirium also may be caused by metabolic and electrolyte derangements, hypoglycemia, hypothermia, and poor analgesia.

DIFFERENTIAL DIAGNOSIS

- Hypoxia
- Hypocarbica or hypercarbia
- Hypotension
- Electrolyte derangement (i.e., hyponatremia, hypercalcemia)
- Hypoglycemia
- Hypothermia
- Acidemia
- Stroke
- Seizure
- Infection
- Central cholinergic syndrome

Immediate Management

- Increase FiO_2 to maintain oxygen saturation.
- Ensure that the patient has an adequate respiratory rate and tidal volume.

Immediate Management (continued)

- Consider drawing an arterial blood gas to measure PaO₂ and PaCO₂.
- Assess the patient's blood pressure and heart rate.
- Verbally reassure and reorient the patient.
- Provide adequate analgesia and anxiolysis if indicated.
- Consider administration of physostigmine 0.5 mg IV if cholinergic syndrome is suspected.
- Restrain patient only if patient or staff safety is at risk.

Diagnostic Studies

- Arterial blood gas analysis
- Fingerstick for blood glucose level
- Laboratory studies: Plasma electrolyte measurements and toxicology screen
- Computed tomography (CT) of brain (if symptoms persist, to rule out acute bleed, stroke)
- If indicated, administer incremental doses of naloxone (40 mcg IV bolus) and/or flumazenil (0.1 mg IV bolus)
- Ensure adequate reversal of neuromuscular blocking agents.

Subsequent Management

- Reassure the agitated or combative patient.
- Correct all metabolic causes and ensure normothermia.
- Consider administration of a butyrophenone (e.g., haldol 2.5 mg IV) to treat agitation without an obvious underlying cause.
- Consider further neurologic consultation and workup (CT scan, magnetic resonance imaging [MRI], electroencephalograph [EEG]) if mental status does not improve.

Risk Factors

- Hypoxia
- Age (children and elderly)
- Organic brain dysfunction
- Mental retardation
- History of substance intoxication or withdrawal

Prevention

Ensure adequate pain control in the postoperative period. Maintain hemodynamic and metabolic stability throughout the anesthetic and into the postoperative period. There is no clear strategy for preventing postoperative excitation and delirium in a susceptible patient. Some studies suggest that the use of dexmedetomidine or

antipsychotics can prevent or reduce the incidence of postoperative delirium.

Special Considerations

- Up to 30%–50% of elderly patients may experience some degree of postoperative confusion, delirium, or cognitive decline. General anesthesia combined with the stress of surgery and pre-existing cognitive abnormalities may all exacerbate this problem. Postoperative cognitive dysfunction may occur immediately in the postanesthesia care unit (PACU) or as late as several days after surgery.

Further Reading

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Neufeld KJ, Leoutsakos JS, Sieber FE, et al. Outcomes of early delirium diagnosis after general anesthesia in the elderly. *Anesth Analg*. 2013; 117(2): 471–478.

Chest Pain

Definition

Pain that may be of cardiac, respiratory, or musculoskeletal etiology. It is frequently described as heavy or pressing, substernal with possible radiation, and is rarely localized. Intensity may vary with respiration.

Presentation

- Acute presentation of substernal chest pain or pressure, with frequent radiation to arm or jaw, described as squeezing pressure.
- Blood pressure changes also may be noted (decreased or elevated).
- May be associated with tachypnea, shortness of breath, dyspnea, cyanosis, or diaphoresis.

Pathophysiology

Postoperative chest pain is common and has a varied etiology. Classic symptoms of chest pain and tightness may be due to myocardial ischemia or infarction. Cardiac chest pain may also radiate to the arms, epigastric area, back, shoulders, neck, or jaw, and may follow skin dermatomes. Gastroesophageal reflux also may be reported as chest pressure or burning pain. Pulmonary thromboembolism may produce sharp, pleuritic pain that varies with respiration.

DIFFERENTIAL DIAGNOSIS

- Myocardial ischemia or infarction (MI)
- Cardiac arrhythmia
- Pericarditis
- Pericardial tamponade
- Pneumothorax
- Pulmonary embolism (PE)
- Pneumoperitoneum
- Gastroesophageal reflux disease
- Esophageal spasm or rupture
- Thoracic aneurysm
- Anxiety

Immediate Management

- Increase FiO_2 to maintain adequate oxygenation.
- Aggressively treat hypotension or hypertension.
- Provide analgesics (morphine) for relief of pain.
- Consider nitroglycerin, either sublingual or as IV infusion (start at 0.5 mcg/kg/min).
- Obtain an electrocardiogram and chest X-ray.
- Initiate a review of surgical and anesthetic procedures.

Diagnostic Studies

- 12-lead electrocardiogram
- Chest X-ray
- Cardiac enzymes

Subsequent Management

- Electrocardiogram (ECG) interpretation should take into account the quality of chest pain, patient cardiac history, baseline ECG, and risk index.
- If a PE is suspected, obtain a high-resolution computed tomography or refer for angiography. Consider duplex ultrasonography of lower extremities to identify the source of a thrombus. Any decision to begin anticoagulation (e.g., a heparin infusion) should be made in conjunction with the surgical service and take into account the risk of bleeding.
- Treat a significant pneumothorax: Needle thoracostomy for life-threatening pneumothorax or request a surgical consultation for insertion of a chest tube if time permits.
- If pericardial tamponade, request an emergency surgical consultation for a pericardiocentesis.

- Consider administering a beta-blocker for control of tachycardia and hypertension (labetalol 5 mg IV or metoprolol 1–2 mg IV).
- Treat hypotension with fluids and/or vasopressors (ephedrine 5 mg IV bolus or phenylephrine 100 mcg IV bolus).
- Consider administering aspirin and/or sublingual nitroglycerin if an MI is suspected.
- If an MI is suspected (especially ST segment elevation MI), obtain a cardiology consultation for possible intervention and revascularization.

Risk Factors

- A cardiac etiology is more likely in the presence of coronary artery disease, peripheral vascular disease, advanced age, male sex, hyperlipidemia, obesity, and diabetes.
- Pulmonary embolism diagnosis is supported in patients with known deep venous thrombosis (DVT), hypercoagulable states, malignancy, advanced age, high risk (e.g., orthopedic) or long procedures and prolonged bed rest.
- A pneumothorax may occur in almost any setting, but should especially be considered after central line placement and surgical procedures involving the chest, neck, and upper abdomen. Also suspect a pneumothorax in any patient with recent trauma.

Prevention

Appropriate preoperative evaluation, cardiac risk stratification, and medical optimization can identify patients at risk for cardiac events. Appropriate DVT prophylaxis (e.g., unfractionated or low-molecular weight heparin) and early ambulation reduces the incidence of deep vein thrombosis and PE. The use of ultrasound may decrease the risk of pneumothorax caused by insertion of a central line.

Special Considerations

- The initial evaluation of chest pain should always first rule out life-threatening conditions, including myocardial ischemia, pneumothorax, and pulmonary thromboembolism. Once those causes have been considered and ruled out, other etiologies (e.g., gastroesophageal reflux) may be considered. Abdominal laparoscopic procedures that induce a pneumoperitoneum can often cause referred pain into the chest, and esophageal pathology may also mimic cardiac pain.

Further Reading

Landesberg G, Beattie WS, Mosseri M, et al. Perioperative myocardial infarction. *Circulation*. 2009; 119(22): 2936–2944.

Hypoxia

Definition

Decreased arterial oxygen content in blood as measured by pulse oximetry or arterial blood gas analysis, usually transient as a result of atelectasis and/or alveolar hypoventilation in the early postoperative period.

Presentation

- $\text{SpO}_2 < 90\%$ or $\text{pO}_2 < 60$ mm Hg
- Patient is frequently lethargic and may be uncooperative or agitated.
- Tachycardia and hypertension may be associated with hypoxia.
- Tissue cyanosis may be seen in severe cases.

Pathophysiology

Hypoxia has many potential etiologies that may be related to comorbidity, the surgical procedure, and anesthesia. Even patients with normal lungs are susceptible to a variety of factors that can cause hypoxia. Factors include respiratory depression from residual anesthesia or neuromuscular blockade, atelectasis, and a ventilation/perfusion mismatch. Potent volatile anesthetics blunt the normal response to hypoxemia.

DIFFERENTIAL DIAGNOSIS

- Hypoventilation due to residual anesthetics
- Hypercarbia
- Atelectasis
- Impaired diffusion (pulmonary edema, pulmonary fibrosis, aspiration)
- Increased oxygen consumption (shivering)
- Pulmonary embolus
- Pneumothorax
- Transfusion-related lung injury
- Acute respiratory distress syndrome
- Mainstem bronchial or esophageal intubation
- Carboxyhemoglobin or methemoglobin (normal PaO_2 but impaired carrying capacity)
- Inadequate FiO_2
- Obstructive sleep apnea

Immediate Management

- Provide supplemental O₂ via nasal cannula or face mask to maintain adequate oxygenation.
- Insert an artificial airway if airway obstruction is present.
- Obtain a serial arterial blood gas analyses to follow PaO₂ and PaCO₂.
- If the patient is severely hypoxemic or respiratory failure is imminent, intubate the trachea and initiate mechanical ventilation.
- Cardiovascular support may be necessary in extreme cases of arterial hypoxia.

Diagnostic Studies

- Chest X-ray
- Arterial blood gas
- Arterial co-oximetry

Subsequent Management

- Continue supplemental O₂.
- Consider intubation and mechanical ventilation for significant hypoxia or hypercarbia.
- Consider naloxone (40 mcg IV bolus), flumazenil (0.1 mg IV bolus), or additional neostigmine and glycopyrrolate to reverse narcotics, benzodiazepines, or neuromuscular blocking agents.
- Provide analgesia if splinting is the cause of hypoventilation.
- Insert a chest tube to decompress a pneumothorax.
- Administer diuretics (furosemide 20 mg IV) to treat volume overload causing pulmonary edema.
- Consider initiating an anticoagulant (e.g., heparin) if PE is suspected. Consult the surgical team before administering anticoagulants.

Risk Factors

- Advanced age
- Obesity
- Pre-existing pulmonary disease (emphysema, fibrosis, pulmonary hypertension)
- Congestive heart failure
- Increased production of carbon dioxide (i.e., malignant hyperthermia, shivering)

Prevention

Prevention of postoperative hypoxia is largely based on appropriate administration of supplemental oxygen in the immediate postoperative period. Appropriate levels of positive end-expiratory pressure and recruitment maneuvers may help to decrease atelectasis and right-to-left shunt. Hypoxia may also be prevented by appropriate postoperative respiratory care to prevent and treat atelectasis.

Special Considerations

Diffusion hypoxia can contribute to postoperative hypoxia after a nitrous oxide anesthetic, and may persist for up to approximately 10 minutes, emphasizing the need for supplemental oxygen immediately after emergence and during patient transport.

Further Reading

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337

Myocardial Ischemia

Definition

Myocardial oxygen supply insufficient to meet metabolic requirements. Ischemia can be caused by decreased regional perfusion or increased oxygen demand.

Presentation

- Chest pain or pressure reported by an awake patient
- ST-segment elevation or depression on ECG
- Possible tachycardia and hypertension or profound hypotension
- Arrhythmias, congestive heart failure, acute mitral regurgitation
- Hypoxia, dyspnea
- Dizziness or syncope
- Timing may occur hours to days after surgery.

Pathophysiology

Etiologies include platelet aggregation, vasoconstriction, and thrombus formation at the site of a plaque in a coronary artery that decreases or interrupts blood flow. In patients with ischemic coronary artery disease, a sudden increase in myocardial oxygen demand (tachycardia) or decrease in oxygen supply (hypotension, hypoxia) can precipitate acute myocardial ischemia and/or infarction.

Immediate Management

- Increase FiO_2 to maintain adequate oxygenation.
- Ensure adequate ventilation.
- Treat pain with narcotics (morphine intravenously [IV]).
- Aggressive fluid resuscitation and/or vasopressors for hypotension.
- Initiate beta-blockers (metoprolol 1–2 mg IV q 5 min or esmolol IV infusion 50 mcg/kg/min to a max of 300 mcg/kg/min) for treatment of tachycardia and hypertension. Titrate to a target heart rate of approximately 60 beats per minute if possible.
- Administer aspirin after consulting with surgeon to determine risk of bleeding.
- Request a cardiology consultation for a possible interventional procedure.

DIFFERENTIAL DIAGNOSIS

- Coronary vasospasm
- Pericarditis
- Aortic dissection
- Pulmonary embolus
- Anxiety

Diagnostic Studies

- 12-Lead electrocardiogram
- Cardiac enzymes (troponin, CPK, CPK-MB)
- Complete blood count, metabolic profile
- Arterial blood gas
- Chest X-ray

Subsequent Management

- Continuous monitoring and serial ECGs and cardiac enzymes to assess for ST segment changes, new Q waves, or conduction defects.
- Echocardiography for new wall motion defects, ejection fraction, valvular dysfunction, or thrombus.
- Administer beta-blockers (e.g., labetalol 5 mg IV or metoprolol 1 mg IV bolus or an esmolol infusion) to control heart rate and blood pressure.
- Consider a nitroglycerin infusion (start at 0.5 mcg/kg/min) for coronary vasodilatation.
- Request a cardiology consultation for possible emergency coronary artery stenting and initiation of anticoagulation and antiplatelet therapy.

Risk Factors

- Known history of coronary artery disease (CAD), previous myocardial infarction, or significant risk factors of CAD
- Major abdominal, thoracic, vascular, or emergency surgeries carry the highest risk of postoperative myocardial infarction.

Prevention

Preoperative optimization of the patient with known or suspected coronary artery disease is essential for prevention of postoperative complications. Maintaining stable hemodynamics helps to ensure adequate myocardial oxygen supply and minimize myocardial demand throughout the perioperative period. With the possible exception of angiotensin converting enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARB) and diuretics, all cardiac medications should generally be continued on the day of surgery.

Special Considerations

Interpretation of ECG changes, including ST segment elevation or depression, must be interpreted taking into account a patient's history and cardiac risk. ST segment depression and chest pain in a low-risk patient rarely indicates myocardial ischemia, but rather a more benign etiology such as anxiety, hyperventilation, and hypokalemia, and generally only requires observation in the PACU. High-risk patients with ST segment changes should undergo an immediate evaluation for myocardial ischemia or infarction. Although monitoring of leads II and V5 detects 80% of ischemic events, a 12-lead ECG should always be performed for verification and evaluation.

Further Reading

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Oliguria/Acute Renal Failure

Definition

Urine output <0.5 mL/kg/h that may signify loss of kidney function due to prerenal, renal, or postrenal etiologies.

Presentation

- Urine output <0.5 mL/kg/h.
- Often associated with a pre-existing renal insufficiency that is exacerbated by an intraoperative event.
- May be associated with electrolyte abnormalities (hyperkalemia), acidosis, and rising blood urea nitrogen and creatinine.

Pathophysiology

Prerenal causes of oliguria include hypovolemia due to bleeding, third-space fluid loss, and inadequate fluid replacement. Preoperative or intraoperative insults, such as radiographic contrast dye, hypotension, sepsis, or exposure to nephrotoxic medications may cause acute tubular necrosis, particularly in a patient with baseline renal insufficiency. Postrenal causes of oliguria include obstruction of the ureters, surgical injury to the ureter, and obstruction of a urinary catheter.

DIFFERENTIAL DIAGNOSIS

- Hypovolemia
- Low cardiac output
- Abdominal hypertension or compartment syndrome
- Acute tubular necrosis
- Nephrotoxic agent exposure
- Ureter obstruction
- Mechanical catheter obstruction.

Immediate Management

- Insert a urinary catheter and evaluate for patency.
- Administer a fluid challenge (10–20 mL/kg crystalloid).
- Discontinue administration of nephrotoxic agents.
- Consider diagnostic administration of a diuretic (furosemide 10–40 mg IV) after ruling out hypovolemia.

Diagnostic Studies

- Basic metabolic panel
- Arterial blood gas
- Chest X-ray
- Urine electrolytes and creatinine

Subsequent Management

- Consider inserting a central venous catheter or noninvasive cardiac output monitor if volume status is unclear.

- Consider starting a vasopressor (norepinephrine 2–4 mcg/min IV or dopamine 2–5 mcg/kg/min IV infusion) for vasodilated states or an inotrope (dobutamine 2–5 mcg/kg/min IV infusion) for low cardiac output to treat hypotension and ensure adequate renal perfusion.
- Treat severe metabolic acidosis and hyperkalemia if present (see pages 126, 144).
- Initiate hemodialysis for worsening renal failure and volume overload refractory to treatment.

Risk Factors

- Chronic renal insufficiency
- Left ventricular dysfunction
- Advanced age
- Significant intraoperative blood loss
- Intraoperative hypotension

Prevention

Careful perioperative fluid balance is important in patients with baseline renal insufficiency. Avoidance of nephrotoxic agents and maintenance of adequate renal perfusion may also be useful in preventing postoperative renal failure.

Special Considerations

- Aortic cross-clamping, severe hypotension, massive transfusion, possible ureteral injury, and other intraoperative events may predispose to postoperative renal failure. Aggressive early rehydration and maintaining an adequate systemic blood pressure are useful in preventing further renal insult.

Further Reading

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Postoperative Hypertension

Definition

Elevated systemic blood pressure, frequently seen with tachycardia.

Presentation

Elevated systolic and/or diastolic blood pressure in postoperative patients. Pain is one of the most common precipitating factors of hypertension commonly associated with tachycardia.

Pathophysiology

Patients with a history of pre-existing hypertension have an exaggerated blood pressure response that is caused by noncompliant vasculature, elevated peripheral vascular tone, and increased endogenous sympathetic nervous system activity. Peripheral arterial and venous constriction is mediated by alpha-adrenergic stimulation causing increased systemic vascular resistance and venous return. Increased contractility and heart rate results from an increase in beta-1 receptor stimulation.

Immediate Management

- Ensure adequate oxygenation and ventilation.
- Administer analgesics (morphine 1–2 mg IV) or anxiolytics (midazolam 1–2 mg) to control pain and anxiety.
- Treat acidosis if present.
- Ensure ability to void or insert a bladder catheter to rule out bladder distention.
- If hypertension persists, consider treatment with intravenous labetalol (5-mg IV bolus), esmolol (10-mg IV bolus or 0.1 mg/kg/min), hydralazine (5-mg IV bolus) or nicardipine (infusion starting at 5 mg/h).

DIFFERENTIAL DIAGNOSIS

- Preoperative essential hypertension
- Postoperative pain and agitation
- Arterial hypoxemia
- Hypercapnea
- Hypervolemia
- Gastric or bladder distention
- Increased intracranial pressure
- Emergence delirium
- Inappropriately small blood pressure cuff or improperly calibrated arterial line

Diagnostic Studies

Initially, none indicated. Further workup should be based on clinical evidence of ruling out an underlying etiology (i.e., acidosis, hypercapnea).

Subsequent Management

- Resume usual antihypertensive medication as soon as possible.
- Persistent hypertension may require intermittent boluses of a beta-blocker (e.g., labetalol), continuous IV infusions (e.g., nicardipine), invasive blood pressure monitoring, and intensive care unit admission.
- Potent vasodilators such as nitroprusside or nitroglycerine are reserved for refractory or life-threatening hypertension.

Risk Factors

- Pre-existing essential hypertension.
- Cessation of antihypertensive medications in the perioperative period.
- Inadequate pain control, either during or after surgery.
- Patients undergoing intracranial procedures.

Prevention

Antihypertensive medications should generally be continued throughout the perioperative period (ACE inhibitors, angiotensin receptor blockers, and diuretics are often held on the morning of surgery) since abrupt withdrawal may precipitate rebound hypertension. Pain should be treated with judicious use of narcotics, regional anesthetic techniques, or other forms of analgesia.

Special Considerations

Postoperative hemodynamic instability in the postanesthesia care unit (PACU) is relatively common, but postoperative systemic hypertension and tachycardia are more predictive of an adverse outcome than are hypotension and bradycardia. Although the exact etiology of hypertension cannot always be found, it is crucial to treat common postoperative causes (i.e., pain, hypoxia) rapidly, and then use pharmacotherapy to restore hemodynamic stability as quickly as possible.

Further Reading

Rodriguez MA, Kumar SK, De Caro M. Hypertensive crisis. *Cardiol Rev.* 2010; 18(2): 102–107.

Postoperative Hypotension

Definition

Decreased systemic blood pressure that may lead to tissue hypoperfusion and ischemia.

Presentation

- Decreased systemic arterial blood pressure associated with either bradycardia or tachycardia.
- Alteration in mental status.
- Organ system failure may be seen after prolonged hypotension (e.g., myocardial infarction, acute renal failure, hepatic ischemia).

Pathophysiology

Systemic hypotension can be categorized as either hypovolemic (decreased preload), cardiogenic (decreased contractility), or distributive (decreased afterload). Hypotension in the PACU is usually caused by decreased preload and ongoing fluid losses (e.g., blood loss). These factors lead to decreased ventricular filling and cardiac output, sympathetic-mediated tachycardia, increased systemic vascular resistance, and venoconstriction. Neuraxial anesthetic techniques may also lead to a loss of sympathetic tone causing hypotension.

Immediate Management

- Ensure adequate oxygenation and ventilation.
- Resuscitate with IV fluids (crystalloid or colloids). Many patients will often respond to a moderate fluid challenge (e.g., incremental boluses of lactated Ringer's solution or normal saline 500 cc IV).
- Support blood pressure with phenylephrine, ephedrine, or epinephrine boluses. Consider beginning an infusion of phenylephrine (0.5–1 mcg/kg/min) or epinephrine (0.03–0.05 mcg/kg/min) in the setting of refractory hypotension.
- Consider placing patient in Trendelenberg position (benefits controversial).

DIFFERENTIAL DIAGNOSIS

- Hypovolemia
- Hemorrhage
- Myocardial ischemia or infarction
- Cardiac arrhythmia
- Cardiac tamponade
- Tension pneumothorax
- Pulmonary embolus
- Anaphylaxis
- Spinal shock
- Sepsis
- Drug-induced (beta-blocker, calcium channel blocker)

Diagnostic Studies

- Chest X-ray if clinical suspicion of pneumothorax.
- Hemoglobin and hematocrit
- Arterial blood gas analysis to determine hematocrit and acid-base status (hematocrit may be unreliable during acute blood loss).
- 12-Lead ECG and/or echocardiogram may be useful if a cardiogenic origin is suspected.

Subsequent Management

Continue supportive care with fluids, blood products, and vasopressors as necessary. Monitor hemodynamics (arterial, central venous pressure) to guide infusion of fluids and drugs. Determine and treat the primary etiology (i.e., myocardial ischemia or pneumothorax).

Risk Factors

- Inadequate replacement of fluids from ongoing fluid losses, including bowel preparation, gastrointestinal losses, or significant bleeding.
- Pre-existing hypertension and cardiac disease.
- Neuraxial anesthetic techniques are often associated with hypotension.

Prevention

Postoperative hypotension is most often prevented by careful intraoperative volume replacement. Blood and fluid losses are often underestimated in many surgical procedures. A fluid load prior to initiating neuraxial anesthesia may help to prevent hypotension. Critically ill patients rely on increased sympathetic tone to maintain normotension and may be more sensitive to anesthetic agents. Administration of ketamine to a catecholamine-depleted patient may cause significant hypotension.

Special Considerations

- Although relative hypovolemia is usually the reason for systemic hypotension, shock resulting from more catastrophic causes must be considered. Significant hemorrhage can develop rapidly in the PACU, often without obvious signs, and must be suspected in any patient with unexplained hypotension. Tension pneumothorax, pulmonary embolus, or pericardial tamponade may occur suddenly and may be lethal if not immediately diagnosed and treated. Anaphylaxis always should be considered as a cause of refractory hypotension.

Further Reading

Cohn SL, Harte B. Postoperative hypertension/hypotension. In: *Perioperative medicine*. London: Springer; 2011:421–430.

Postoperative Nausea and Vomiting

Definition

Significant nausea or vomiting experienced either in the PACU or after discharge is a common complication.

Presentation

- Range of symptoms from mild nausea to retching and vomiting in the postoperative period.
- Severe vomiting increases intra-abdominal pressure and may disrupt abdominal or inguinal suture lines.
- Increased risk of gastric aspiration, especially when airway protective reflexes are not completely intact.
- Increased sympathetic nervous system response, producing hypertension and tachycardia.

Pathophysiology

Postoperative nausea and vomiting (PONV) is the result of multiple of perioperative factors such as fasting, autonomic imbalance, pain, and anesthetic effects on chemotactic centers. The emetic centers are located in the lateral reticular formation of the medulla. Pharmacologic treatment aims to target specific receptors, including dopaminergic, histaminergic, cholinergic, substance P, and serotonergic.

Immediate Management

- Increase FiO_2 to ensure adequate oxygenation. Exclude potentially serious causes of PONV (i.e., impending shock, increased intracranial pressure).
- Ensure adequate hydration with intravenous crystalloids.
- Administer medication for rescue (i.e., ondansetron 4 mg IV). Do not repeat the same class of agents used for prophylaxis unless a prolonged benefit was previously noted (>6 hours).

DIFFERENTIAL DIAGNOSIS

- Anesthetics (narcotic, inhalational agents)
- Pain
- Anxiety
- Hypoxia

- Hypotension
- Hypoglycemia
- Increased intracranial pressure
- Gastric bleeding
- Bowel obstruction

Diagnostic Studies

- Clinical diagnosis
- Request additional diagnostic studies as clinically indicated to rule out other more severe etiologies.

Subsequent Management

- Continue to treat severe PONV with multimodal therapy.
- Phenothiazines (promethazine 25 mg IV or prochlorperazine 10 mg IV) are good second-line agents.
- Droperidol (0.625 mg IV) is highly effective but carries a black-box warning for QTc prolongation.
- Propofol (10–20 mg) may also be beneficial for refractory nausea.

347

Risk Factors

- Female sex
- Nonsmoking status
- Prior history of motion sickness or PONV
- Laparoscopic surgery
- Strabismus and middle ear surgery
- Opioid-based and nitrous oxide anesthetic
- Without prophylaxis, 10%–80% of patients may experience postoperative nausea and vomiting (PONV) after a volatile anesthetic technique.

Prevention

Prophylaxis of PONV is often more effective than rescue therapy. Patients who have a higher risk of developing PONV should receive double- or triple-agent prophylaxis. Prophylaxis in patients at very low risk may not be indicated.

Special Considerations

Patients with a history of protracted PONV, despite adequate prophylactic therapy, may benefit from an alteration in anesthetic technique in addition to the usual prophylactic treatment. Regional anesthesia techniques reduce the incidence of PONV significantly. When a general anesthetic is required, limited use of narcotics, volatile anesthetics, and nitrous oxide may be possible with a total intravenous anesthetic technique.

Further Reading

Gan TJ, Diemunsch P, Hib AS, et al. Consensus guidelines for the management of postoperative nausea and vomiting. *Anesth Analg*. 2014; 118(1): 85–113.

Prolonged Neurologic Impairment after Regional Anesthesia

Definition

Prolonged neurological impairment after regional anesthesia that may include prolonged paralysis or sensory deficit after a neuraxial anesthesia technique. The deficit may be caused by the anesthetic, its contaminants, surgical procedure, positioning, or exacerbation of a pre-existing condition.

Presentation

- Prolonged motor and sensory blockade that is denser or longer in duration than expected.
- Sensory deficits such as numbness and paresthesias.

Pathophysiology

The most serious neurologic complication of neuraxial anesthesia is paraplegia due to ischemia, infection, or drug toxicity. Spinal cord ischemia or infarction may be due to arterial hypotension or compression of the cord by an expanding epidural hematoma or abscess. Neurotoxicity can also occur as a result of either intrathecal local anesthetics or preservatives.

DIFFERENTIAL DIAGNOSIS

- Spinal/epidural hematoma
- Spinal/epidural abscess (not acute)
- Neuropathy due to positioning (i.e., lithotomy position)
- Toxicity of anesthetic or additive introduced into the intrathecal space
- Cauda equina syndrome
- Longer than expected effect of anesthetic agents

Immediate Management

- Neurologic examination to evaluate extent of residual effect.
- Schedule an emergency MRI of the spinal cord to evaluate for hematoma or other pathology.
- Request an emergency neurosurgical consultation.

Diagnostic Studies

Emergency MRI of spinal cord after an intrathecal or epidural block.

Subsequent Management

- Emergency surgical decompression if evidence of hematoma or abscess.
- Other than surgical decompression, further treatment of significant neurologic injury is largely supportive.
- Most transient paresthesias resolve over about 6 months.
- Numerous case reports show unexplained prolonged block with no known nerve damage (i.e., clonidine patch prolonging blockade).

Risk Factors for a Hematoma

- Leukemia, blood dyscrasias, thrombocytopenia
- Antiplatelet therapy or anticoagulation
- Traumatic or difficult needle placement

Prevention

Although it is impossible to prevent all neurologic complications of spinal and epidural anesthetics, proper patient selection is important in limiting the potential of catastrophic complications. Avoid regional anesthesia in patients who are treated with antiplatelet agents (aspirin alone is usually acceptable) and therapeutic anticoagulation. Regional anesthesia should also be avoided in patients who have an ongoing significant infection or who are severely immunocompromised.

Special Considerations

An association between paresthesias and radiating pain on injection of local anesthetics has been suggested. Although redirecting the needle may be safe if a paresthesia develops during placement, consider stopping the procedure or relocating the needle if a paresthesia occurs during local anesthetic injection.

Further Reading

Horlocker TT. Regional anaesthesia in the patient receiving antithrombotic and antiplatelet therapy. *Br J Anaesthes*. 2011; 107(Suppl 1): i96–i106.

Respiratory Depression or Failure

Definition

Impaired ventilation or oxygenation or loss of airway patency due to a variety of mechanical, hemodynamic, and pharmacologic factors.

Presentation

- Airway obstruction and loss of pharyngeal muscle tone
- Decreased minute ventilation and possible hypoxia
- Excessive sedation and somnolence
- Residual neuromuscular blockade with muscle weakness

Pathophysiology

The residual effects of intravenous and inhalational anesthetics blunt the normal responses to both hypercarbia and hypoxemia. Benzodiazepines and narcotics act synergistically to further decrease respiratory drive, and can cause upper airway obstruction. Residual neuromuscular blockade may cause further compromise of the upper airway. Pharyngeal muscles normally contract with the diaphragm to open the airway during inspiration, but this function is often impaired in the sedated patient, causing airway obstruction and respiratory depression.

DIFFERENTIAL DIAGNOSIS

- Residual anesthetics (benzodiazepines, opioids, volatile anesthetics)
- Residual neuromuscular blockers
- Upper airway obstruction, edema, or hematoma
- Laryngospasm
- Bronchospasm
- Pulmonary edema
- Pulmonary embolus
- Pneumothorax
- Obstructive sleep apnea

Immediate Management

- Increase FiO_2 to maintain adequate oxygenation.
- Perform a jaw thrust or insert an oral, nasal, or laryngeal mask airway.
- Intubate the trachea for respiratory failure or for significant airway obstruction that does not immediately resolve with the preceding interventions.

Diagnostic Studies

- Chest X-ray
- Arterial blood gas

- Assess the patient for the presence of residual neuromuscular blockade.

Subsequent Management

- Reverse residual opioids or benzodiazepines with incremental doses of the appropriate antagonist (i.e., naloxone 40 mcg IV or flumazenil 0.1 mg IV).
- Reverse residual neuromuscular blocking agents.
- Administer beta-2 agonists for treatment of bronchospasm or reactive airway disease.
- Initiate continuous positive airway pressure (CPAP).
- Intubate the trachea and initiate mechanical ventilation.

Risk Factors

- Advanced age
- Obesity
- Chronic obstructive pulmonary disease
- Severe asthma
- Obstructive sleep apnea

351

Prevention

The synergistic effects of respiratory depressants (e.g., residual anesthetic agents, opioids) are a common cause of hypoventilation. Titrate sedatives and opioids carefully, especially in patients who are prone to postoperative respiratory failure.

Special Considerations

- Hypoventilation and airway obstruction may be seen after discontinuing noxious stimuli (i.e., extubation or infiltration of a wound with local anesthetic solution). Slow titration of naloxone (40 mcg IV incremental doses) can reverse opioid-induced respiratory depression without affecting analgesia. An overdose of naloxone causes severe pain, tachycardia, hypertension, and possibly myocardial ischemia. Consider the possibility of a hematoma obstructing the airway in a patient who has just undergone neck surgery.

Further Reading

Murphy GS, Szokol JW, Marymont JH, Greenberg SB, Avram MJ, Vender JS. Residual neuromuscular blockade and critical respiratory events in the postanesthesia care unit. *Anesthes Analges*. 2008; 107(1): 130–137.

Severe Postoperative Pain

Definition

Postoperative pain is a complex physiological response to tissue trauma, visceral distention, and ongoing disease processes. The goal of postoperative pain management is to provide subjective comfort while inhibiting trauma-induced nociceptive signals in order to blunt autonomic and somatic reflex responses to pain.

Presentation

- Constant pain near the surgical site, burning or aching in nature.
- Frequent acute exacerbation of pain with normal postoperative activities such as coughing, ambulating, and dressing changes.
- Hypertension, tachycardia, myocardial ischemia, increased oxygen consumption, and increased sympathetic tone may be present.

Pathophysiology

Nociception involves the recognition and transmission of noxious stimuli via afferent sensory nerves through the dorsal horn of the spinal cord to the contralateral cortex via the thalamus. Modulation of this signal may occur at any level of the pathway with medications such as opioids, NSAIDs, cyclooxygenase-2 inhibitors (COX-2), and local anesthetics.

Immediate Management

- Evaluate the quality and level of pain using a visual analog scale or similar method.
- If the pain is severe and refractory to appropriate management, re-evaluate the patient and notify the surgical team.
- For severe postoperative pain, opioids are the mainstay of therapy.
- Administer incremental doses of opioids (e.g., morphine 1–2 mg IV or hydromorphone 0.2 mg IV) until patient appears comfortable. A pain score of 3–4 is usually acceptable.
- Consider administering acetaminophen 1 g IV as an adjuvant to opioids for improved analgesia.
- Provide supplemental O₂ to ensure adequate oxygenation in case opioids cause hypoventilation.

DIFFERENTIAL DIAGNOSIS

- Inadequate analgesic regimen (inadequate or infrequent dosing)

- Wound complications (infection, hematoma, nerve injury)
- Chronic pain disorder
- Drug-seeking behavior

Diagnostic Studies

Clinical diagnosis

Subsequent Management

- Intrathecal or epidural administration of local anesthetics and opioids may provide superior pain relief in many abdominal or lower-extremity orthopedic procedures.
- Nonsteroidal anti-inflammatory drugs and COX-2 inhibitors have no hemodynamic effects and do not cause respiratory depression, but decrease levels of inflammatory mediators at the site of tissue injury. Cyclooxygenase-2 agents do not affect platelet function.
- Consider switching opioids if pain control is inadequate (i.e., morphine to hydromorphone or meperidine).
- Consider patient-controlled analgesia (PCA) once pain is at an acceptable level and the patient is comfortable.
- Transition pain medications to oral route as soon as feasible.

353

Risk Factors

- Male sex
- Preoperative pain
- Prior history of poor pain management
- Coexisting medical conditions (substance abuse, withdrawal, anxiety)
- Opioid addiction

Prevention

Pre-emptive analgesic therapy may significantly help in controlling postoperative pain in many patients. Local and regional anesthetic techniques can be used to decrease the intensity and duration of postoperative pain.

Special Considerations

- In addition to patient comfort, adequate control of pain may have a further benefit in improving clinical outcome by preventing myocardial ischemia or infarction, impaired wound healing, atelectasis, and thromboembolic events. Postoperative pain is usually a self-limiting condition with progressive improvement over a relatively short period of time. The severity of postoperative pain has been shown to predict chronic pain.

Further Reading

Apfelbaum JL, Ashburn MA, Connis RT, et al. Practice guidelines for acute pain management in the perioperative setting: an updated report by the American Society of Anesthesiologists Task Force on Acute Pain Management. *Anesthesiology* 2012; 116(2): 248.

Stroke

Definition

Development of a new acute focal neurological deficit due to either an ischemic or embolic event in the brain, diagnosed either immediately after emergence or early in the postoperative period.

Presentation

- Weakness or paralysis in one or more extremities
- Cranial nerve deficits and/or dysarthria
- Severe confusion and altered mental status
- Blurred vision or diplopia

Pathophysiology

A stroke may occur as the result of an occlusion of a cerebral artery and subsequent infarct of brain tissue due to profound hypotension, a thrombotic event, or an embolism (e.g., air or fat). Embolic strokes may be of cardiac origin. Hemorrhagic strokes due to aneurysm or arteriovenous malformation are also possible, but far less common in the perioperative setting.

DIFFERENTIAL DIAGNOSIS

- Thrombo-embolic stroke
- Ischemic stroke
- Hemorrhagic stroke
- Intracerebral hemorrhage
- Gas embolism (through a patent foramen ovale)
- Seizure

Immediate Management

- Ensure adequate oxygenation and ventilation.
- Ensure hemodynamic stability, using vasoactive drug infusions as needed to maintain an adequate systemic arterial blood pressure (systolic blood pressure of 120–140 mm Hg is adequate for most patients).
- Request an emergency CT of brain without contrast.
- Request an emergency neurology consultation and call a “stroke code.”

Diagnostic Studies

- Clinical diagnosis
- Brain CT to rule out bleeding
- Possible MRI of brain

Subsequent Management

- Consider inserting an arterial catheter to facilitate continuous hemodynamic monitoring.
- Consider initiating tissue plasminogen activator (t-PA) or antiplatelet therapy if there is no surgical contraindication.
- Request a neurosurgical or neurology consultation for possible endovascular intervention.

Risk Factors

- Recent stroke
- Transient ischemic attack (TIA) or amaurosis fugax
- Cardiac, carotid, or neurosurgical procedure
- Atrial fibrillation
- Significant hypotension

355

Prevention

Preoperative screening for significant carotid disease or risk of cardiogenic embolus may be of benefit. Careful surgical technique during cardiac and carotid surgery is also crucial to prevent embolism of arterial plaques, thrombus, or air entry. Avoidance of hypotension in at-risk patients is also important to reduce the risk of stroke due to hypoperfusion and ischemia.

Special Considerations

- Many patients exhibit slurred speech or behavior that under other circumstances might indicate a primary neurologic event while emerging from a potent volatile anesthetic. Close observation is necessary to assure that patients return to their expected level of functioning prior to discharge from the PACU.

Further Reading

Macellari F, Paciaroni M, Agnelli G, Caso V. Perioperative stroke risk in non-vascular surgery. *Cerebrovasc Dis.* 2012; 34(3): 175–181.

Chapter 13

Regional Anesthesia Complications

Nikhil Bhatnagar

Complete Spinal Anesthesia	358
Epidural Abscess	360
Epidural Hematoma	363
Globe Injury	366
Local Anesthetic Systemic Toxicity	367
Peripheral Nerve Injury	370

Complete Spinal Anesthesia

Definition

Inadvertent spread of local anesthetic to cervical and brain stem regions after a regional block resulting in loss of consciousness, cardiovascular instability, and respiratory failure.

Presentation

- Initial symptoms are consistent with a high spinal and include nausea and vomiting, hypotension, and bradycardia.
- Symptoms then progress to upper extremity anesthesia and respiratory distress.
- Eventually the patient may lose consciousness.
- All of these symptoms may occur simultaneously or in rapid succession.

Pathophysiology

A complete spinal anesthetic causes significant venous and arterial dilation, decreasing preload and afterload, and resulting in hypotension. Bradycardia is caused by blockade of the T1 to T4 spinal roots, which contain the cardiac accelerator fibers. Blockade of the C3 to C5 nerve roots results in phrenic nerve paralysis and respiratory arrest. When the local anesthetic reaches the brain stem, further hemodynamic and respiratory compromise occurs because many of the respiratory and vasomotor centers are located in the brain stem.

DIFFERENTIAL DIAGNOSIS

- Local anesthetic systemic toxicity (LAST)
- Vasovagal reaction
- High spinal anesthesia
- Excessive sedation
- Anaphylactic reaction

Immediate Management

- Stop injecting local anesthetic.
- Place patient in reverse Trendelenberg position to prevent a further increase in block height. If a total spinal anesthetic is already present, position the patient with his or her head down to increase venous return to the heart.
- Increase FiO_2 to 100%.
- If necessary, intubate the trachea and initiate mechanical ventilation.

Immediate Management (continued)

- If the patient is conscious but requires general anesthesia for airway management, etomidate or ketamine should be used to minimize hemodynamic instability.
- Support the blood pressure with fluid and vasoconstrictors (e.g., epinephrine 10- to 20-mcg bolus or 0.1–0.2 mcg/kg/min, ephedrine 5-mg bolus, and phenylephrine 100-mcg bolus or 1 mcg/kg/min) Note: Phenylephrine may exacerbate bradycardia.
- Immediately begin advanced cardiac life support if cardiac arrest occurs!

Diagnostic Studies

- This is a clinical diagnosis based on the symptoms occurring after a spinal anesthetic.
- A computed tomography (CT) or magnetic resonance image (MRI) of the brain and spinal cord only if symptoms persist after several hours or the patient is not arousable.

Subsequent Management

- If patient has persistent hypotension and respiratory depression, cancel the surgical procedure and transfer the patient to the intensive care unit (ICU) for further monitoring.
- Symptoms of total spinal usually resolve in 90–120 minutes if bupivacaine is injected. Shorter-acting anesthetics will have quicker resolution.
- If the total spinal anesthetic may have been caused by a malpositioned epidural catheter, aspirate the catheter. If cerebrospinal fluid (CSF) is drawn into the syringe, remove the catheter.

Risk Factors

- High volume or total dose of local anesthetic used for spinal or epidural anesthesia.
- Placing a patient into the supine or head-down position too rapidly after initiating an epidural or spinal anesthetic.
- Using a high lumbar or low thoracic site for an epidural or spinal anesthetic.
- Increased intraabdominal pressure: Epidural vein engorgement decreases epidural volume. Pregnant and obese patients are at higher risk for this complication.
- Elderly patients are at higher risk because of decreased compliance of the epidural space.

Prevention

Minimize total dose and volume of local anesthetic during spinal and epidural anesthesia. Aspirate an epidural catheter to prevent local anesthetic from being injected into the intrathecal space. A test dose of 3 cc of 1.5% lidocaine with 1:200,000 epinephrine should be injected before dosing an epidural catheter. Local anesthetic should be administered through an epidural catheter slowly and in divided doses. After initiating an epidural or spinal anesthetic, the patient should remain sitting for at least 30 seconds to avoid cephalad spread.

Special Considerations

- Spinals and epidural anesthetics are not the only regional techniques that can result in a total spinal anesthetic. This complication has been reported after interscalene, retrobulbar, peribulbar, paravertebral, intercostal, and lumbar plexus blocks.

Further Reading

- 360 Caplan R, Ward R, Posner K, Cheyney F. Unexpected cardiac arrest during spinal anesthesia: a closed claims analysis of predisposing factors. *Anesthesiology*. 1988; 68(1): 5–11.
- Carpenter R, Caplan R, Brown D, Stephenson C, Wu R. Incidence and risk factors for side effects of spinal anesthesia. *Anesthesiology*. 1992; 76(6): 906–916.

Epidural Abscess

Definition

A collection of purulent matter (neutrophils, bacteria, and necrotic cells) within the epidural space that causes compression of the spinal cord, leading to subsequent neurologic sequella and possibly meningitis or sepsis.

Presentation

- Radicular back pain is the most common presenting symptom.
- Erythema and fluctuance at insertion site.
- Fever (only occurs in one-third of patients but is usually the initial symptom)
- Leukocytosis
- Meningitis (nuchal rigidity and stiffness). This is relatively uncommon but can occur with cervical epidurals.
- Neurologic symptoms (sensory, motor, and bladder/bowel incontinence). This is a relatively late symptom.

- Symptoms usually occur 4–7 days after needle insertion, but this is highly variable.
- Classic triad of fever, back pain, and neurologic signs only occurs 11% of the time.
- Sepsis is a late and ominous finding of an untreated epidural abscess.

Pathophysiology

The most common mechanism is direct inoculation of the epidural space with skin or respiratory flora (*S. Aureus*, *S. epidermidis*, and *P. aeruginosa*). The infection causes an inflammatory reaction and creation of purulent matter in the posterior epidural space causing a mass effect that produces neurologic sequelae. If the infection spreads to the blood stream, signs of systemic infection will occur with eventual progression to sepsis.

DIFFERENTIAL DIAGNOSIS

- Epidural hematoma
- Direct needle insertion and injection of local anesthetic into the spinal cord
- Transient neurologic symptoms
- Meningitis
- Spinal headache

Immediate Management

- Obtain immediate MRI or CT imaging of the spine if signs or symptoms of an epidural abscess are present.
- If imaging studies demonstrate an epidural abscess, transfer the patient to the ICU.
- Request emergency neurosurgical and infectious disease consultations.
- If neither neurologic symptoms nor signs of sepsis are present, surgery may be delayed and the patient can be managed on antibiotics. Blood cultures and CT-guided cultures guide management.
- Initial broad spectrum antibiotic coverage includes vancomycin, a third-generation cephalosporin, and gram-negative coverage (e.g., gentamicin) and is narrowed after a diagnosis is made.
- If the patient shows signs of neurologic deterioration, request an emergency neurosurgical consultation for posterior decompression.

Diagnostic Studies

- Magnetic resonance image of spine

- Computed tomography of spine
- Complete blood count with differential
- Blood and epidural cultures

Subsequent Management

- Antibiotic therapy is managed by an infectious disease specialist, and is usually 3–4 weeks in duration.
- If the diagnosis made early enough, there may be no neurologic sequelae. Late diagnosis may lead to sepsis and permanent neurologic impairment.
- The amount of time from symptom onset to surgical decompression determines neurologic outcome.

Risk Factors

- Immunocompromised patients, including diabetics, patients on chemotherapy and radiation, patients who are treated with immunosuppressive drugs, and patients with human immunodeficiency virus (HIV).
- Patients who are septic or who have an active infection.
- Patients who require an epidural catheter for long period of time, usually >3 days.
- Lack of aseptic technique during insertion of an epidural catheter or injection of medications.

Prevention

The best prophylaxis is strict aseptic technique, including hand washing and wearing a hat, a mask, and sterile gloves during insertion. The insertion site should be prepped with either chlorhexadine or a betadine solution and a sterile drape applied. Immunocompromised patients and those with signs of active infection warrant consideration of the infection risk before a neuraxial technique is performed. If an indwelling catheter is in place, it should be examined every day for signs of infection. Limit the duration of indwelling catheters to the minimum amount necessary.

Special Considerations

- Unlike epidural hematomas, which occur in the hospital and usually within the first 48 hours, an epidural abscess often occurs a week or more after needle insertion. Therefore, all patients who have neuraxial anesthesia should be given information about symptoms of epidural abscess before discharge.

Further Reading

- Grewal S, Hocking G, Wildsmith JMW. Epidural abscesses: review article. *Br J Anaesthes*. 2006; 96 (3): 292–302.
- Wang L, Hauerberg J, Schmidt J. Incidence of spinal epidural abscess after epidural analgesia: a national 1-year survey. *Anesthesiology*. 1999; 91(6): 1928–1936.
- Mackenzie AR, Laing RBS, Smith CC, Kaar GF, Smith FW. Spinal epidural abscess: the importance of early diagnostics and treatment. *J Neurol Neurosurg Psychiatry*. 1998; 65(2): 209–212.

Epidural Hematoma

Definition

Penetration or injury of the epidural vessels resulting in a collection of blood that leads to compression of the spinal cord and subsequent neurologic sequelae.

Presentation

- Radicular back pain
- Bladder dysfunction
- Sensory dysfunction that outlasts the expected duration of the block or increases in intensity when no additional anesthetic has been injected.
- Motor dysfunction that outlasts the local block or increases in intensity when no additional local anesthetic has been injected.
- There is no specific sequence of events that makes the diagnosis. Any of these symptoms can occur in isolation, simultaneously, or at different times.

Pathophysiology

Spinal hematoma is most commonly caused by disruption of the epidural venous plexus. The expanding hematoma creates a mass effect because the cross-sectional area of the spinal canal is fixed. Continued expansion displaces the CSF and causes further compression of the arteries, veins, and eventually the spinal cord, ultimately causing progressive spinal cord ischemia. Injury can be either transient or permanent.

DIFFERENTIAL DIAGNOSIS

- Epidural or spinal abscess
- Needle insertion and local anesthetic injection into the spinal cord
- Transient neurologic symptoms

Immediate Management

- Discontinue local anesthetic infusions through the epidural catheter.
- Symptoms of cord compression mandate an immediate MRI or CT of the spine.
- Request an emergency neurosurgical consultation if radiographic evidence of a hematoma is observed.
- If patient is at high risk for a spinal hematoma, neurologic checks should occur every 2 hours, preferably in an ICU.
- Symptom progression from radicular back pain to paralysis can occur very rapidly, usually only 14 hours after initial onset of symptoms. Any delay in surgical decompression will worsen the prognosis, so rapid diagnosis and intervention are imperative.

Diagnostic Studies

- Computed tomography of spine (fast but less optimal)
- Magnetic resonance image of spine (slower but gold standard)

Subsequent Management

- A rapidly deteriorating patient should be evaluated immediately by the neurosurgical service for surgical decompression.
- No more than 8 hours should elapse from symptom onset to neurosurgical decompression.
- If the patient is not rapidly deteriorating or the hematoma is small, a conservative course of close observation, serial neurologic exams, and MRIs may be appropriate.

Risk Factors

- Coagulopathy, including patients with kidney failure, liver failure, pre-eclamptic patients, or patients who have disseminated intravascular coagulation. As a general rule, international normalized ratio (INR) should be <1.5 or platelets $>100,000$.
- Treatment with anticoagulant, antiplatelet, or fibrinolytic drugs. Examples include heparin, platelet inhibitors (e.g., clopidogrel), thrombin inhibitors (e.g., dabigatran), and others.
- Multiple attempts at regional or a bloody puncture.
- Epidural catheter placement and removal is associated with higher probability of epidural hematoma than is a single shot epidural or spinal anesthetic.
- Patients with spinal stenosis, spinal tumors, or other space occupying lesions have higher chances of having clinically significant epidural hematomas because of smaller spinal canal diameters.

Prevention

The most important preventive measure is a focused history and physical, especially in high-risk patients. A careful history of antithrombotic medication use should be elicited, with special attention to when these drugs were stopped. Laboratory studies should include a platelet count and INR. Note: Some drugs, such as low-molecular weight heparin, cannot be monitored with conventional laboratory studies. Newer laboratory studies (e.g., functional platelet levels, factor X assays, and thromboelastogram) provide more comprehensive information about coagulation status, especially in patients treated with newer anticoagulants.

Special Considerations

The sensory and motor deficits that occur during a local anesthetic block can mimic the symptoms of a spinal hematoma. Epidural infusions should be periodically discontinued in order to assess motor and sensory function. Heavy sedation may mask symptoms, and developmentally delayed patients may not be able to express symptoms or cooperate with an examination. If a patient develops a coagulopathy or is given an anticoagulant when an epidural catheter is in place, discontinue local anesthetic administration, discontinue anticoagulation, and follow serial coagulation studies. The patient should have frequent neurologic checks and the epidural catheter should be removed only when all values have normalized.

Patients treated with nonsteroidal anti-inflammatory drugs, aspirin, or subcutaneous heparin (<5000 U TID) may be considered for regional anesthesia. The American Society of Regional Anesthesia has published guidelines for the use of regional anesthetics in patients receiving antithrombotic therapy.

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Globe Injury

Definition

Incidental globe penetration (entry) or globe perforation (entry and exit) can occur with accidental needle puncture during either retrobulbar or peribulbar block

Presentation

- Extreme intraocular pain on injection
- Loss of visual acuity
- Dark red reflex
- Decrease in intraocular pressure

Pathophysiology

Introduction of needle into globe results in disruption of structures within the globe such as the retina and retinal vessels. Injection of local anesthetic can also lead to chemical injury. Ocular pressure is only rarely increased if local anesthetic solution is injected directly into the globe.

366

DIFFERENTIAL DIAGNOSIS

- Retrobulbar hemorrhage
- Injection into optic nerve
- Retinal detachment

Immediate Management

- Stop the injection immediately and withdraw the needle.
- Request an emergency consultation from an ophthalmologist.
- Cancel the surgical procedure.

Diagnostic Studies

- Fundoscopic examination
- Ultrasound examination. Note: This is especially helpful during the block procedure if globe penetration is suspected.

Subsequent Management

- Cancel or defer the surgical procedure. Decreased intraocular pressure can further predispose the patient to retinal hemorrhage and retinal detachment.
- Management is determined by the ophthalmologist and depends upon the extent of the injury. Therapy may include transscleral cryotherapy, laser retinopexy, vitrectomy, silicone oil tamponade, and scleral buckling.

Risk Factors

- Excessively sedated patients
- Myopic patients with axial lengths >26 mm
- Blunt needles tend to cause more trauma and that trauma is more difficult to treat as compared to sharp needles.

Prevention

Retrobulbar techniques are associated with a much higher chance of globe perforation than are peribulbar techniques. Experienced practitioners are less likely to cause globe perforation while performing an ocular block. Avoid excessive sedation during ocular blocks.

Special Considerations

- Anesthesiologists and ophthalmologists have identical complication rates. A fundoscopic examination always should be performed after this block in order to detect this complication more quickly and accurately. Delay in diagnosis can lead to irreparable vision loss.

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Local Anesthetic Systemic Toxicity

Definition

Local anesthetic overdose that results in rapidly progressive central nervous system (CNS) and cardiac failure.

Presentation

- Central nervous system excitation: Prodromal symptoms such as agitation, confusion, metallic taste, and auditory changes only precede seizures 20% of the time. Seizures are usually the primary manifestation of LAST. Central nervous system depression and coma occur after seizures.
- Cardiac depression: Hypertension, tachycardia, and ventricular arrhythmia precede cardiovascular depression such as bradycardia, decreased ventricular conduction and contractility, and asystole.

- CV symptoms usually occur after CNS symptoms. More potent local anesthetics have a low cardiovascular collapse (CC) to CNS ratio, which causes cardiac symptoms to occur simultaneously or even precede CNS symptoms. Bupivacaine is the classic local anesthetic with a low CC:CNS ratio.
- Symptoms of LAST typically occur within the first minute after injection. This usually indicates intravascular injection. However, it is not uncommon for symptoms to occur several minutes or even hours later in cases in which there is a partial intravascular injection or a perineural/epidural catheter in place.

Pathophysiology

Local anesthetic's primary mechanism of action is to bind to the intracellular side of the sodium channel, blocking the propagation of action potentials. Local anesthetics also block potassium channels and calcium channels at clinically relevant concentrations, which explains some of the side effects (e.g., decreased contractility and widening QRS complex). When the blood concentration of local anesthetic reaches the toxic range, to neurons in the CNS and cardiac nerve cells (bundle branch blocks and widening of QRS) are bound, inhibiting their function. At extremely high concentrations (lidocaine plasma concentration 5–10 mcg/mL and bupivacaine plasma concentration of 0.5–5 mcg/mL) local anesthetics will bind to sodium channels on the myocytes causing cardiac arrest.

DIFFERENTIAL DIAGNOSIS

- Total spinal anesthesia
- Vasovagal reaction
- Severe hypotension
- Anaphylactic reaction

Immediate Management

- Immediately stop injecting local anesthetic.
- Resuscitate the patient. Administer supplemental oxygen or intubate the trachea patient is unable to protect their airway.
- If patient is having seizures consider administration of a benzodiazepine (lorazepam 1 mg or midazolam 2 mg in an adult). Avoid propofol because it may cause further myocardial depression and hypotension.
- If patient is in cardiac arrest initiate advanced cardiovascular life support (ACLS). Do not administer lidocaine because this will exacerbate LAST. Administer epinephrine at much lower doses than the 1-mg dose typically given to adults (typically 1 mcg/kg) because high-dose epinephrine has been linked

Immediate Management (continued)

to worse outcomes in local anesthetic cardiac arrests. (The myocardium is especially susceptible to arrhythmias.)

- Follow serial ABGs. Acidosis, hypoxemia, and hypercarbia all exacerbate the effects of LAST.
- Administer lipids as early as possible. Administer a 1.5-mL/kg bolus of 20% lipid emulsion 1.5 mL/kg; continue as an infusion at 0.25 mL/kg/min over the next 30–60 minutes.

Diagnostic Studies

- LAST is usually a clinical diagnosis.
- Echocardiogram to look at structure and function of heart. Especially valuable when cardiac manifestations of LAST.
- Serum local anesthetic levels. These are only really valuable if an epidural or nerve block catheter is placed and the patient is having vague prodromal LAST symptoms on the floor. These labs should not be drawn if there is an acute emergency.
- Consider an EEG if seizures are intractable

Subsequent Management

- Lipid emulsion therapy can be repeated after the initial bolus. Maximum dose is 12 mL/kg.
- Myocardial depression may last for 80 to 90 minutes with LAST. Do not discontinue resuscitation efforts.
- If lipid emulsion therapy fails, consider open cardiac massage or cardiopulmonary bypass.

Risk Factors

- Potent local anesthetics with low CC:CNS ratios (e.g., bupivacaine). Avoid 0.75% bupivacaine because this has been associated with the most LAST complications.
- Nerve blocks in areas that have inherently high rates of intravascular absorption. The rate of absorption from high to low are as follows intrapleural > intercostal > lumbar plexus > paravertebral > caudal > epidural > brachial plexus > femoral > sciatic > transverse abdominus plane block > subcutaneous > intra-articular > spinal.
- Pregnant patients have engorged epidural veins and thus higher rate of absorption. Progesterone may also lower the threshold for LAST.
- Patients with systemic diseases such as cardiac, hepatic, and renal failure because of decreased LA metabolism and lower cardiac reserve if LAST occurs.

Risk Factors (*continued*)

- Patients at extreme age ranges. The very young do not have fully developed renal and hepatic systems and are more prone to LA toxicity. The very old have decreased hepatic and renal function. By age 80, functioning nephrons have decreased by 50%.

Prevention

Use the lowest volume and concentration (especially of bupivacaine) of local anesthetic that will achieve the desired result. Inject slowly and in divided doses and aspirate for blood frequently. Inject 5 mL of local anesthetic, waiting 30 seconds between doses. Epinephrine containing solutions (1:200,000) can be used as a marker for intravascular injection. Epinephrine also decreases systemic absorption of local anesthetic by 33%.

Special Considerations

- Heavy sedation may mask the prodromal effects preceding LAST. The timing and sequence of LAST symptoms is highly variable, so one should have a low threshold for diagnosis. Although LAST typically occurs within a minute of injection, the patient should be monitored for at least half an hour. Lipid emulsion, along with intubating equipment and emergency drugs, always should be immediately available when performing a regional anesthetic. Lipid emulsions are thought to bind local anesthetics in the blood stream, mitigating their toxic effects.

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Peripheral Nerve Injury**Definition**

Mechanical, pharmacologic, or ischemic disruption of one or more structural components of a nerve, causing either a transient or permanent deficit.

Presentation

- Sensory changes include anesthesia, paresthesia, hyperalgesia, and allodynia along the nerve distribution.
- Motor changes include paresis or paralysis along the nerve distribution.
- Autonomic changes include sudomotor dysfunction (which may be indicated by increased or decreased sweating in the affected limb), swelling, and temperature changes along nerve distribution (complex regional pain syndrome).
- Less severe nerve injury tends to be associated with sensory deficits; motor and autonomic changes are more likely with more severe injury.
- Injury usually occurs immediately after needle insertion and injection. Injection pressure is higher than usual and patients usually, but not always, complain of paresthesias.

Pathophysiology

The most likely mechanisms of injury is mechanical injury, ischemia, or toxicity that occurs when the needle is inserted directly into the fascicle and local anesthetic is injected under high pressures. Needle insertion itself does not usually cause lasting injury.

DIFFERENTIAL DIAGNOSIS

- Stroke
- Position injury (much more common etiology than regional anesthesia)
- Cervical or lumbar disc disease
- Muscle or tendon injury

Immediate Management

- If patient has a paresthesia during needle insertion, stop and evaluate the patient. If paresthesia is persistent, abandon the procedure and observe. If paresthesia abates, reposition the needle.
- Do not inject local anesthetic if injection is painful or if high pressure is required to inject the anesthetic. Reposition the needle.
- If patient has a complete or progressive motor and sensory deficit after local anesthetic has worn off, request an immediate surgical consultation.

Diagnostic Studies

- This is a clinical diagnosis based upon the sequence of events during the procedure

- Electromyography and nerve conduction study
- Magnetic resonance imaging to rule out spinal injury
- Computed tomography to rule out spinal injury

Subsequent Management

- Incomplete or minor deficits after local anesthetic has worn off require observation and serial EMG and NCS.
- A baseline EMG/NCS demonstrates only that nerve injury has occurred, and not the type of injury. It is not necessary to order a baseline EMG/NCS.
- Wallerian degeneration will be complete after 10–14 days if there is axonal loss. Therefore, an NCS should be able to distinguish between neurapraxia with demyelination or actual axonal loss. This information is extremely valuable to determine the course of the injury because neurapraxia almost always resolves quickly, whereas axonal loss may be permanent or take significantly longer to resolve.
- Electromyography has a more protracted time course. Fasciculations and spontaneous electrical activity indicate nerve injury that is 2–5 weeks old.
- A follow-up EMG/NCS should be done at 3 and 6 months.
- If the injury has not improved after 2–5 months, consider a neurosurgical evaluation. Spontaneous improvement in symptoms after 18–24 months is rare because Schwann cell tubes collapse.
- Gabapentin and tricyclic antidepressants may ameliorate neuropathic pain.

Risk Factors

- Patients with medical conditions that compromise the nerve (e.g., diabetes, Marie Charcot Tooth syndrome, or patients who have received chemotherapy).
- Pre-existing nerve injury in the distribution being blocked. (Injured nerves are more susceptible to local anesthetic toxicity—the *double crush phenomenon*).
- Prolonged tourniquet application.
- Regional anesthetics performed on sedated or anesthetized patients. Consider risk/benefit in mentally challenged patients or pediatric patients, who may be unable to tolerate a nerve block while awake.
- Injecting under high pressure
- High-concentration local anesthetic
- Use of vasoconstrictor such as epinephrine, which reduces perineural blood flow

Prevention

- Avoid injection of local anesthetic under high pressure.
- Avoid performing blocks in oversedated or anesthetized patients. The patient should be able to complain of paresthesias or pain during the procedure.
- Proceed with caution, and if possible, avoid regional anesthesia in patients with medical conditions that predispose to nerve injury or who have a pre-existing nerve injury.
- Avoid performing a nerve block at the same level as the tourniquet. The tourniquet can cause significant perineural ischemia and increase the risk of a double crush injury.
- A short beveled needle should always be used when doing a nerve block in order to reduce the risk of penetrating the nerve.

Special Considerations

- Peripheral nerve injury is an exceedingly rare complication, with an incidence of 0.4 per 1000. Nerve injuries are more likely to occur as a result of malpositioning than from regional anesthesia. The vast majority of injuries are transient sensory injuries.
- Ultrasound guidance and nerve stimulation are equally safe, but local anesthetic should not be injected if stimulation is occurring <0.2 mA.
- The most benign type of injury is *neurapraxia* (focal demyelination with no disruption of nerve elements). Full recovery usually occurs within 2–12 weeks. In *limited axonal loss*, relatively few axons have been destroyed, and the fascicle and perineurium are intact. Full recovery usually occurs in between 2 and 4 months. *Intermediate axonal loss* involves the destruction of several axons and the interruption of a few fascicles. Patients usually recover within 4 to 6 months. *Severe axonal loss* involves interruption of the majority of fascicles with intact epineurium and stromal elements. There is significant sensorimotor loss and recovery, which is usually incomplete, takes between 6 and 18 months. Complete nerve discontinuity is the most severe injury; the nerve is effectively transected and the epineurium and all stromal elements are disrupted. Recovery will not occur without surgical intervention.
- Symptom duration is highly variable and depends on the extent of neuronal injury. Minor injuries are self-limited and complete recovery usually takes place within weeks. Recovery from more severe injury may take as long as 18 months to 2 years and may only be partial. In the most severe cases, the nerve injury could progress to complex regional pain syndrome.

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Equipment Problems

René R.P.M. Hagenouw and James B. Eisenkraft

Breathing Circuit Malfunction: Low Pressure Condition after Induction of Anesthesia	376
Electric Power Failure	379
Equipment Malfunction before Induction of Anesthesia	382
Computer Network Failure	383
Oxygen Pipeline Failure	385
Ventilator Failure after Induction of Anesthesia	388

Each operating room is equipped with a combination of devices that include an anesthesia machine or workstation, physiologic monitors, infusion pumps, an anesthesia information management system (AIMS) and clinical information systems. These may be grouped together in different combinations as an Anesthesia Machine or they may be used separately in the care of a patient undergoing a surgical, diagnostic, or therapeutic procedure. Because all of these devices are interconnected, failure in one may affect others that are connected to the same patient. This chapter discusses the management of malfunctioning patient care equipment.

Breathing Circuit Malfunction: Low Pressure Condition after Induction of Anesthesia

Definition

Failure to generate or sustain a positive pressure in the breathing circuit that is sufficient to ventilate the patient's lungs.

Presentation

- Breathing system fails pre-use checkout.
- During spontaneous ventilation, reservoir bag empties; FiO_2 is less than intended; low FiO_2 alarm sounds.
- During bag-assisted ventilation, bag empties easily when squeezed but the circuit is not pressurized and the lungs do not inflate.
- During positive pressure ventilation with a ventilator:
 - Breathing system low pressure alarm is annunciated during case if properly set.
 - Capnogram is abnormal or absent (apnea) leading to alarm.
 - Ventilator standing bellows fails to fill or sinks to the bottom of its housing.
 - Spirometry alarm for low tidal volume (TV) or minute volume (MV) is annunciated.

Malfunction Details

The breathing system must be gas tight to ensure that the patient receives the correct gas mixture and that positive pressure is transmitted to the lungs during inspiration.

DIFFERENTIAL DIAGNOSIS

- Leak in the anesthesia machine low-pressure system
- Breathing system disconnection or misconnection

- Breakage of, or leakage from, a breathing system component that may be obvious or concealed
- Leak around airway management device (tracheal tube, laryngeal mask airway [LMA])
- Gas leak from the bronchial tree (e.g., bronchopleural fistula)

Immediate Management

- If the patient's lungs cannot be ventilated, immediately disconnect the breathing circuit and attempt ventilation with a self-inflating manual ventilation device (SIMVD) connected to a source of oxygen.
- Inspect anesthesia workstation and breathing system for an obvious leak. Correct if possible. Call for help to repair the faulty component while continuing to care for the patient.
- If the leak is small, increase the fresh gas flow (FGF) to compensate while the cause is determined.
- Differentiate between a problem with the ventilator or breathing circuit up to the Y-piece and a problem with the elbow, tracheal tube, or LMA by switching the circuit to the reservoir bag, disconnecting the Y-piece from the swivel connector, blocking the Y-piece with a finger, closing the pop-off valve, and squeezing the bag to determine if the circuit can be pressurized.
- If the problem is with the equipment on the patient side, check swivel and elbow connectors and the tracheal tube pilot balloon or LMA seating. If necessary, prepare to change the airway device.
- If the problem is on the workstation side and increasing the fresh gas flow compensates for the gas leak, monitor the ventilation closely and be ready to replace the ventilator.
- If the leak is on the workstation side and cannot be compensated for by increasing FGF, disconnect the patient from the breathing system and ventilate the lungs with a SIMVD connected to an oxygen source if available (otherwise use room air). Call for help and replace the ventilator.
- Maintain anesthesia with intravenous agents.

Diagnostic Studies

- A difference in inspired and expired tidal volumes indicates that there is a leak in the breathing system that may be proximal (e.g., a hole/defect in the circuit tubing) or distal to the Y-connector (e.g., tracheal tube cuff leak, poorly seated LMA). If the breathing system incorporates a flow sensor between the Y-piece and the patient's airway (e.g.,

D-Lite flow sensor used in GE ADU workstation or optional in GE Aisys Carestation), a leak distal to the flow sensor can be distinguished from a leak in the circuit between the workstation and the flow sensor.

- Verify that the set ventilator TV agrees with inspired and expired TVs.

Subsequent Management

- If the source of the leak cannot be determined, replace the anesthesia breathing system and/or ventilator as soon as feasible.
- Do not start another anesthetic until the equipment has been replaced or repaired.
- Refer to authorized service personnel.

Risk Factors

- Failure to perform the manufacturer's recommended pre-use checkout procedures.
- Failure to correctly assemble breathing system components. Note: Be especially careful when using a new model and especially when switching from one manufacturer's equipment to that of another.

Prevention

- Educate personnel in the correct operation of the workstation, including pre-use checkout.
- Use all available breathing system monitors and alarms. Early detection and intervention decrease the risk of an adverse outcome.

Special Considerations

- Any connection can become disconnected or misconnected. Always re-check the breathing circuit and ventilator when re-entering an operating room after leaving for any reason.

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Electric Power Failure

Definition

- Loss of electrical power. May be caused by an electric utility company power outage, or failure within the facility (e.g., severing a cable during construction).
- Most facilities have backup generators that turn on automatically when a utility company supply failure occurs, but a period of several seconds to a minute may elapse before the backup generator supplies power after a failure. Some facilities in the hospital (e.g., the operating room) may have uninterruptible power supplies. These backup systems may fail during a fire or during natural disasters such as storms, earthquakes, or acts of terrorism.

Presentation

- Room lighting fails.
- Electrical devices connected to line power turn off unless they have a battery backup or are connected to an uninterruptible power supply (UPS).
- Some or all anesthesia devices may shut down, depending on whether they have internal backup batteries.
- Local telephone and paging systems may shut down.
- Cardiopulmonary bypass machine, cell saver, electrocautery, robotic systems, and other life support or essential equipment will fail.
- If the failure is caused by a community power outage and emergency generators are activated, electrical power should be restored within a short period of time. During the transition, some electronic equipment (e.g., computers) may power down and take time to reset.

DIFFERENTIAL DIAGNOSIS

- Failure of a supply outside the facility (e.g., excessive power demand during extremely hot or cold weather, weather-related

power line failure, natural disasters, fire, earthquake, construction work).

- Unannounced construction work or a failure of individual circuits in the operating room (OR) (e.g., turned off accidentally, or by a tripped circuit breaker) causing shutdown of one or more electrical devices.

Immediate Management

- Set up emergency portable lighting (flashlight, laryngoscope light, emergency flashlights mounted on wall in most ORs).
- Call for help. If the OR telephone system fails, use a cell phone.
- Ensure that the power loss is not due to patient or personnel electrocution.
- If an electrical panel is in the room, check for tripped circuit breakers.
- Ensure that all essential electrically powered equipment is connected to emergency power outlets in the OR; these are usually identifiable by a red wall plate.
- Check that oxygen pipeline gas supply and suction are functioning; otherwise, follow the protocol for oxygen pipeline failure.
- Check that the ventilator is functioning and that the patient's lungs are being ventilated. If not, switch to manual ventilation by breathing system reservoir bag or self-inflating resuscitation bag.
- Discuss the situation with the surgeon/proceduralist. Abort the procedure if possible. If possible, awaken the patient.
- Re-establish patient monitoring. Obtain a battery powered transport monitor for electrocardiogram (ECG), noninvasive blood pressure (NIBP), capnography, pulse oximetry.
- The ECG can be monitored using the monitoring mode of a defibrillator
- Use a precordial or esophageal stethoscope to monitor ventilation
- Obtain a manual BP cuff and sphygmomanometer to monitor blood pressure. Palpate the pulse.
- Consider using a colorimetric CO₂ detector or a battery powered semi-quantitative CO₂ mainstream analyzer (e.g., Nellcor Easy Cap by Covidien, Masimo EMMA™ Emergency Capnometer) to monitor CO₂.
- Electronic workstations have a backup battery that will supply power for 30–45 minutes. Conserve battery power

Immediate Management (continued)

by switching off the ventilator and manually ventilate using the reservoir bag. Adjust the screen brightness control to the lowest possible setting.

- On workstations that normally have electronic display of gas flows, revert to 100% O₂ from backup rotameter (Dräger Apollo®, Dräger Fabius® GS, GE S5/ADU®). If using a GE Aisys® workstation, switch to the “Alternate Oxygen” flowmeter.
- Reassign personnel to provide manual power (e.g., hand-ventilating the patient’s lungs cranking the cardiopulmonary bypass machine).
- Postpone elective surgical procedures until the electrical supply has been restored.

Diagnostic Studies

Refer to authorized engineering and service personnel.

Risk Factors

- Failure to regularly check emergency backup power supply systems and to maintain or repair as required. (Anesthesia personnel should always be represented on hospital equipment and facilities committees.)
- Unannounced construction or maintenance in the vicinity of the institution.
- Unannounced construction or maintenance in the vicinity of the ORs.

381

Prevention

- Regularly test the emergency power supply to ensure that it starts without delay and that automatic transfer occurs as soon as the power is stabilized. Service and repair as needed.
- Provide adequate warning to all staff of any planned interruption of electrical power supply (e.g., maintenance or construction).
- Educate OR personnel about the power supply to the OR, how to react to a power supply failure, and about electrical safety.
- During checkout of the anesthesia devices, note the charge status of the backup battery.
- Regularly check all anesthesia devices to ensure that the batteries are charged and can maintain their charge. Replace backup batteries as necessary.
- Regularly check that battery-powered lighting/emergency flashlights are functional, can be easily found in a dark room, and charging in each anesthetizing location.

- Consider obtaining uninterruptible power supply (UPS) units for electrically powered devices (e.g., OR computers and anesthesia information management systems) that do not have a backup battery.

Special Considerations

- Most facilities have backup generators that activate automatically during a power outage, and some areas within the hospital (e.g., the operating room) may be supplied with an uninterruptible power supply. However, these backup systems may fail during a fire or during natural disasters such as storms, earthquakes, terrorism act, and so on.

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Equipment Malfunction before Induction of Anesthesia

Definition

Failure of one or more devices (e.g., ventilator, patient monitors, AIMS, Clinical Information Systems, TEE) that is deemed essential for the case fails to power up, does not successfully complete self-test or does not work as expected.

Immediate Management

- If a critical piece of equipment (e.g., anesthesia workstation, physiologic monitor) has failed, do not induce anesthesia.
- Verify that the device is connected to a source of electricity and other required sources (e.g., gas, vacuum). Check for continuity of hoses and power cords.
- Check switches and knobs. Equipment malfunctions are frequently caused by incorrect control settings.

Immediate Management (*continued*)

- If a computer (e.g., AIMS, clinical information system) is inoperative, attempt to reset using the front panel switch or by cycling the power.
- Contact biomedical engineering or an anesthesia technician to repair or replace faulty equipment. If equipment cannot be replaced or repaired, move the patient to another operating room.
- Depending on institutional policy it may be possible to proceed with surgery if some equipment is not functioning (e.g., a nonfunctional AIMS may be replaced by a paper-based anesthesia chart).

Subsequent Management

- The affected operating room should be taken out of service until the equipment has been repaired or replaced.
- Examine the operating room carefully for factors that may have caused the initial problem (e.g., a tripped circuit breaker that interrupts power to an anesthesia workstation)

Risk Factors

- Improperly maintained equipment. Moving equipment from one location to another may cause cables or hoses to become partially disconnected. This may not be obvious unless they are examined carefully.
- Inadequate training on complex equipment, including anesthesia workstations.

Prevention

- Test all equipment before the patient enters the room.

Computer Network Failure**Definition**

- Failure of the OR computer network. May be restricted to one OR, the entire OR complex, or the entire facility.

Presentation

- The hospital information system is not responding.
- The AIMS may or may not be charting measurements for ventilator and physiologic monitors, depending upon how it is connected to the other anesthesia devices.

- The AIMS cannot connect to the hospital information system to retrieve patient data or lab results, or cannot address network printers.
- The duration of the outage depends upon its cause, the structure of the network, and the level of redundancy.

Immediate Management

- Determine whether the AIMS is acquiring data from the workstation and physiologic monitors. If not, revert to paper record keeping.
- Check for loose or unplugged data cables. Consider rebooting computers that do not have a life support function.
- Report the network failure. Many hospital IT departments do not have equipment that automatically signals a network failure.

Subsequent Management

- If the data network outage affects a hospital-wide electronic health record system, consider postponing surgery until alternate methods of retrieving patient information are available.

Risk Factors

- Routine data center maintenance.
- Faulty network equipment.
- Unannounced construction or maintenance in the vicinity of the institution.
- Unannounced construction or maintenance in the vicinity of the ORs.

Special Considerations

- Periodically review “downtime” procedures.
- A plan should be in place to retrieve laboratory results and other critical information if the network should malfunction.
- Paper anesthesia records should be available for emergency use in all anesthetizing locations.

Further Reading

Fujii S, Moriwaki K, Sanuki M, et al. Loss of anesthesia records during network failure of anesthesia management information system: a case of malfunction of backup system. *Masui*. 2014; 63: 575–577.

Oxygen Pipeline Failure

Definition

Oxygen pipeline supply pressure to the workstation is either absent or below the minimum oxygen pressure required for normal function of the anesthesia workstation.

Presentation

- Workstation fails pre-use checkout.
- Low oxygen supply pressure alarm is annunciated in workstation.
- If there is a problem with the central supply, a low oxygen pressure alarm may sound in the OR control center.
- O₂ flow at main and auxiliary flow meters decreases or stops; the oxygen flush valve fails.
- Other gases supplied to machine (N₂O, Heliox, possibly air) stop flowing.
- Oxygen-powered pneumatic ventilator stops working; apnea alarms sound (i.e., low pressure, low tidal volume, absent capnogram).

Malfunction Details

- Pipeline supply of oxygen at 50 psig or 3–5 Bar(g) enters the workstation intermediate pressure system and supplies the following:
 - 55 psig diameter indexed safety system (DISS) oxygen outlet (to drive jet ventilator, venturi suction device)
 - Pneumatically powered ventilator
 - Auxiliary flowmeter (e.g., for nasal cannula)
 - O₂ flush
 - Main oxygen flow meter
 - Alternate oxygen flow meter (GE Aisys® workstation)
 - Low oxygen pressure alarm
 - Fail-safe mechanism
- Failure of the central oxygen supply or the pipeline to the OR, pipeline system closed off or obstructed (e.g., by debris)
- Obstruction of the hose connecting the oxygen wall outlet to the workstation.

DIFFERENTIAL DIAGNOSIS

- Leak in anesthesia machine oxygen system
- Obstruction or kinking of hose between wall oxygen outlet and workstation

- Debris in the pipeline system or a malfunctioning wall oxygen outlet
- Shutoff valve outside the OR is in the OFF position.
- Unannounced maintenance of pipeline system
- Failure of connection between bulk oxygen storage vessel and pipeline system.

Immediate Management

- Confirm loss of pipeline oxygen pressure by checking the pipeline supply pressure gauge on workstation. Check O₂ flush operation.
- Open the reserve O₂ cylinder on the gas machine or ventilator.
- Minimize oxygen use.
 - Use the lowest O₂ flow possible (closed-circuit technique if possible).
 - Switch off the pneumatic ventilator; use spontaneous or manual ventilation. (Most anesthesia ventilators use oxygen as the driving gas.)
 - Ensure that the auxiliary oxygen flow is off.
- Announce the failure. Call for help and for additional backup oxygen tanks.
- Alert the surgeon, personnel in other ORs, and the engineering department.
- If workstation has pipeline supply of air and the patient will tolerate a lower FiO₂, consider decreasing FiO₂ to conserve oxygen.
- If the ventilator is driven by O₂ and use of compressed air is not possible, hand-ventilate with room air using a self-inflating resuscitation bag and maintain anesthesia using intravenous agents.
- Postpone elective surgical procedures until the pipeline oxygen supply has been restored and an adequate supply of backup oxygen cylinders is available.

Diagnostic Studies

- Use the oxygen analyzer to ensure that the patient is receiving an adequate FiO₂.
- Ensure that the patient is maintaining an adequate SpO₂.
- Refer to authorized engineering and service personnel.

Risk Factors

- Failure to perform manufacturer recommended pre-use checkout procedures
- Unannounced construction in the vicinity of the OR

Risk Factors (continued)

- Unannounced pipeline maintenance
- Filling of bulk oxygen storage vessel by unqualified personnel.

Prevention

- Teach all anesthesia providers how to prepare, operate, and troubleshoot the anesthesia workstation.
- Check for normal pipeline pressure before starting an anesthetic, and periodically throughout the day.
- Check all hose connections between wall and workstation for tightness, leaks, and condition.

Special Considerations

- Ensure that backup tank of oxygen on the anesthesia gas machine is tightly secured in the hanger yoke before starting an anesthetic.
- Ensure that the tank is full, and then turn it off. If the tank is left open, oxygen may leak out between the tank and the hanger yoke, and if the pipeline pressure drops below 45 psig, oxygen will be drawn from the tank. In either case, the backup tank will slowly become depleted.
- Ensure that all anesthesia personnel have received instruction and practiced changing the oxygen tank on the anesthesia gas machine.
- Consider having a backup supply of E size O₂ tanks that are filled to 3000 psig and will deliver 1000 liters of gaseous O₂, and that have a regulator and DISS oxygen connection that can provide oxygen at 50 psig. If the pipeline supply fails, the oxygen hose can be disconnected from the wall outlet and connected to the 55 psig DISS connector on the tank. These tanks can be stored in a central location within the operating room and distributed as necessary during a pipeline failure.

Further Reading

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Ventilator Failure after Induction of Anesthesia

Definition

- The ventilator does not deliver the desired volume of inspired gas to the patient's lungs.

Presentation

- Ventilator bellows movement is abnormal (irregular, sticking) or absent.
- Ventilator does produce normal sounds during inspiration.
- One or more ventilator alarms are activated (e.g., low/high tidal volume, minute ventilation; low, high, or continuing pressure).
- Physiologic monitors may indicate inadequate ventilation (low or high end-tidal carbon dioxide; low SpO₂).
- Clinical signs of hypoventilation/apnea include failure of the chest to move normally during ventilation, absence of breath sounds on auscultation, and a patient who is attempting to breathe spontaneously.

Pathophysiology

- Bellows ventilators are powered by a compressed gas (usually oxygen but some may use compressed air) while piston ventilators are driven by an electric motor. Both designs use electronic controls that control the mechanical parts (i.e., the piston, or valves to control the compressed gas). An electronics failure in either type of ventilator or failure of the gas supply to bellows ventilators will render the system inoperative.
- Ventilator operation may not be intuitively obvious. An anesthesia provider who is unfamiliar with the system may unintentionally select an incorrect mode, resulting in failure to ventilate.
- The ventilator must be connected correctly to a properly configured breathing system.
- Fresh gas flow may be absent or inadequate to overcome a small leak in the breathing system.
- Alteration in thoracic compliance during the procedure may necessitate a change in ventilator parameters.
- Breathing system or airway obstruction
- Patient "fighting" the ventilator (e.g., light anesthesia, inadequate neuromuscular blockade)
- Failure of the pneumatic and/or electrical power supply to the ventilator
- Failure of a ventilator component (may be mechanical, electronic, computer control system, or pneumatic)

- Failure of breathing system flow sensor. Properly functioning flow sensors are required for the normal operation of most modern ventilators.
- Failure of the decoupling valve in a ventilator that uses fresh gas decoupling.

DIFFERENTIAL DIAGNOSIS

- Ventilator turned off or set to an incorrect mode.
- Failure of the ventilator pressure relief valve (stuck open and leaking or stuck closed, causing a high pressure condition in breathing system)
- Obstructed inspiratory side of the breathing circuit

Immediate Management

- Immediately switch to manual ventilation with the reservoir bag, fill the circuit using the oxygen flush, and attempt to ventilate the patient. If this is successful, continue manual ventilation while troubleshooting.
- If the breathing circuit cannot be filled by operating the oxygen flush, ventilate the patient's lungs using a SIMVD (e.g., Ambu) bag. If a source of compressed oxygen or air is available, a Bain circuit may be used. In the absence of back-up ventilation equipment, mouth-to-tracheal tube ventilation may be necessary.
- Call for assistance. Continue to monitor the patient and ask an anesthesia technician or another anesthesia provider to troubleshoot and correct the problem.
- Check the breathing circuit for disconnections, misconnections, and other sites of gas leakage (e.g., perforated tubing). Check that the pipeline gas supply is functioning; if not, switch to backup cylinder supplies.
- If the circuit fills from the oxygen flush or flowmeters, but the patient's lungs cannot be ventilated, switch to a SIMVD, a Bain circuit, or other alternative ventilation system. Look for a possible obstruction on the inspiratory side of the circle system (e.g., a stuck inspiratory unidirectional valve, occluded inspiratory limb). (Note: Plastic packaging may completely or partially cover the end of a breathing hose.)
- If unable to ventilate the patient's lungs with any backup system, consider an airway obstruction (e.g., kinked tracheal tube, herniated cuff, mucus plug or foreign matter in the tube or bronchial tree, endobronchial intubation, bronchospasm, tension pneumothorax).

Immediate Management (*continued*)

- If potent volatile anesthetics cannot be administered, maintain anesthesia using a total intravenous anesthesia (TIVA) technique.
- Ensure that ventilation and oxygenation are maintained using the backup system if necessary, until the failure has been identified and the problem corrected.
- If the problem cannot be corrected by an anesthesia caregiver or anesthesia technician, the workstation should be withdrawn from clinical use until it is repaired by manufacturer-authorized service personnel.

Diagnostic Studies

- Ensure that the patient is receiving adequate FiO_2 using an oxygen analyzer.
- Ensure that the patient has an adequate SpO_2 .
- Refer to authorized engineering and service personnel.

Risk Factors

- Failure to perform manufacturer's recommended pre-use checkout procedures
- Failure to maintain or service the anesthesia gas machine according to manufacturer's recommendations
- Unfamiliarity with the operation of the workstation/ventilator

Prevention

- All anesthesia providers should receive training in the use of the anesthesia workstation.
- Some newer workstations use fresh gas decoupling to prevent changes in tidal volume when fresh gas flow rate, respiratory rate, or I:E ratio are altered. The user must understand the differences between this design and that of the traditional systems.
- Perform proper pre-use checkout of the workstation and ventilator
- Properly maintain and service all equipment according to manufacturer's recommendations.
- Confirm correct assembly of the breathing circuit, function of unidirectional valves, and ability to ventilate a test lung (i.e., a reservoir bag) connected at the Y-piece of the circle system in both manual/bag and automatic/ventilator modes, and proper function of bag/ventilator selector switch.

- Before starting an anesthetic, ensure that an alternative means to ventilate the patient's lungs (e.g., a self-inflating manual ventilation device) is immediately available and functioning.

Special Considerations

- Standard E cylinders on the anesthesia machine are filled to approximately 2200 psig and deliver approximately 684 L of gaseous O₂ at 1 atmosphere pressure.
- To estimate the time remaining to emptying of an E cylinder at a given flow rate, use the formula:

$$\begin{aligned} \text{Time remaining (hours)} \\ = \text{Pressure (psig)} / (200 \times \text{O}_2 \text{ flow rate (l/min)}) (\text{U.S.}) \end{aligned}$$

e.g., If pressure = 1000 psig and flow rate is 10 L/min:

$$1000 / (200 \times 10 \text{ L/min}) = 1000 / 2000 = 0.5 \text{ hours}$$

$$\begin{aligned} \text{Time remaining (hours)} \\ = \text{Pressure (Bar)} / (30 \times \text{O}_2 \text{ flow rate (l/min)}) (\text{Metric}) \end{aligned}$$

Further Reading

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Chapter 15

Procedures

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Anesthetic Implications of Pacemakers	394
Awake Intubation	396
Cricothyroidotomy	399
Double Lumen Endotracheal Tube	401
Femoral Vein Catheter	403
Intubating Laryngeal Masks	405
One Lung Ventilation	407
Retrograde Intubation	409
Transcutaneous Pacing	411
Transport of a Critically Ill Patient	413
Transvenous Pacing	416
Ultrasound-Guided Central Venous Access	418

Anesthetic Implications of Pacemakers

Although implantation of cardiac pacemakers was originally indicated for symptomatic bradycardia and high-grade atrioventricular block, indications have expanded to include improving cardiac function via synchronized biventricular pacing in patients with dilated cardiomyopathy. Pacemakers can be single chamber (atrial or ventricular) or dual chamber (atrial and ventricular). Leads can be either unipolar or bipolar. Most cardiologists now implant a bipolar lead, which has a smaller distance between the anode and cathode. This decreases the risk of electromagnetic interference.

Pacemakers can be programmed into three modes, *asynchronous*, *single cardiac chamber demand*, or *dual cardiac chamber sequential demand*. In the asynchronous mode, the pacer delivers pulses at preset time intervals and is not affected by intrinsic cardiac activity. Asynchronous mode is preferred to avoid a disruption in pacing if unipolar electrocautery will be used during surgery. In the demand mode, the pacer delivers pulses only when no cardiac complex is sensed in a set amount of time, as may occur with bradycardia. Demand mode is susceptible to possible inhibition caused by electrocautery interference.

The most commonly used pacemaker code is comprised of five letters (Table 15.1). The first letter position represents the cardiac chamber paced (A = atrium, V = ventricle, and D = dual chamber, O = none). The second letter signifies the cardiac chamber sensed (A, V, D, or O). The third letter represents the response (O = none, I = inhibited, T = triggered, or D = dual). The fourth letter includes rate modulation = R. The fifth letter represents anti-dysrhythmia function (A, V, D, or O).

Table 15.1 Generic Pacemaker Code (NBG) NASPE (now known as the Heart Rhythm Society)/BPEG Revised (2002)

Position I	Position II	Position III	Position IV	Position V
O = none	O = none	O = none	O = none	O = none
A = atrium	A = atrium	I = inhibited	R = rate modulation	A = atrium
V = ventricle	V = ventricle	T = triggered		V = ventricle
D = dual (A + V)	D = dual (A + V)	D = dual (T + I)		D = dual (A + V)

Source: Reprinted with permission from Bernstein AD, et al. The Revised NASPE/BPEG Generic Code for antibradycardia, adaptive-rate, and multisite pacing. *PACE* 2002; 25: 260–264.

Before Surgery

- Determine whether the patient has a pacemaker.
- Establish whether the device is a pacemaker or an implantable cardiac defibrillator.
- Determine the device manufacturer: The patient should have a card with the relevant information. If this is unavailable, attempt to contact the patient's cardiologist or primary care physician. (This is not true anymore.)
- Determine whether the patient is pacemaker dependent either by patient history, obtaining medical records, or via device interrogation. Pacing spikes on an ECG may not be sufficient to make this diagnosis. (Are pacing spikes seen on a resting ECG?)
- If time permits, interrogate the pacemaker to determine its settings.
- In an emergency and the preceding information is not available, an ECG can be performed looking for pacing spikes. A chest x-ray can help determine where and how many chambers are paced.

During Surgery

- Apply standard physiologic monitors as determined by patient status and ASA Guidelines.
- If unipolar electrocautery will be used during a surgery on a pacemaker-dependent patient, switch the device to asynchronous mode.
- If the patient has an ICD, turn off the antitachyarrhythmia function and place defibrillator pads on the patient until the device is reactivated.
- Placing a magnet on top of the majority of pacemakers will cause the device to revert to asynchronous mode. (Note: Depending upon the manufacturer and programmed settings, the rate may indicate remaining battery life.) Placing a magnet on top of an ICD will deactivate the defibrillator portion but will not change the programmed pacer function. A device representative must be present to reprogram the pacemaker.
- Removing the magnet should restore pacemaker and ICD functions to their original settings. Interrogate the device after surgery.
- If pacing is affected by electrocautery and a magnet is not on the patient, place a magnet on top of the pacemaker to convert to asynchronous mode. If unipolar electrocautery must be used, the surgeons should use short bursts of energy on the "cutting" setting whenever possible. The electrocautery return pad should be located as far from the pacemaker as feasible. Surgeons should use bipolar electrocautery whenever possible.

- If a hemodynamically significant tachyarrhythmia develops, defibrillate the patient.
- Intraoperative myocardial ischemia and high blood levels of local anesthetics may elevate the electrophysiologic capture threshold, causing loss of capture. If this occurs, transcutaneous pacing may be required.

Events associated with pacemaker interference include:

- Hyperventilation (lowers serum potassium level)
- Shivering
- Electrocautery
- Nerve stimulators
 - Neurophysiologic monitoring
 - Muscle fasciculations
 - Large tidal volumes
 - Magnetic resonance imaging
 - Radiofrequency ablation
 - Electroconvulsive therapy

After Surgery

- Remove the magnet to restore the baseline pacing mode.
- Monitor the patient until the device is interrogated.
- If an ICD was deactivated through a programming change, a defibrillator and pads must be immediately available until this function is restored.

Further Reading

Stone ME, Apinis A. Current perioperative management of the patient with a cardiac rhythm management device. *Semin Cardiothorac Vasc Anesthes*. 2009; 13(1): 31–43.

Awake Intubation

Definition

Awake intubation is the securing of the patient airway prior to the induction of general anesthesia. Intubation with a fiberoptic bronchoscope (FOB) is the most commonly used technique, but any technique can be used to insert an endotracheal tube (ETT) if the patient is properly prepared.

Indications

Consider awake intubation when airway evaluation reveals a potential difficult intubation and one of the following:

- A potential cannot-ventilate by face mask or supraglottic airway (SGA)
- A potential aspiration risk
- A patient who would suffer significant oxyhemoglobin desaturation if the airway cannot be secured rapidly

Contraindications

- Patient's inability to cooperate
- Allergy to local anesthetics

Complications

- Failed intubation

Equipment Checklist

- Parenteral antisialogogue (e.g., glycopyrolate 0.2–0.4 mg)
- A nasal vasoconstriction agent (e.g., oxymetazoline or phenylephrine)
- Local anesthetic (Lidocaine 2% and 4% solutions or viscous preparations are most commonly used.)
- Several 5-mL syringes and small-gauge needles (e.g., 25 G) as well as several plastic catheters from large-bore intravenous catheters (needle removed)
- Cotton pads or swabs
- Alcohol, chlorhexidine, povidone iodine solution for skin preparation
- McGill forceps

Technique

Many techniques have been described. Topical and/or invasive blocks can be used as described in the following. The anesthesia provider should limit the total dose of lidocaine to 600 mg.

- Administer an antisialogogue before any topical blocks are applied to the oral cavity.
- Consider judicious use of light sedation for the procedure.

Many different sedative agents can be used, but use the least number of individual agents (usually not more than two). Administer sedation in small increments to keep a patient cooperative and responsive. Reversal agents should be readily available. Suitable agents include fentanyl (25 mcg IV incremental doses), midazolam (0.5 mg IV incremental doses), and dexmedetomidine (0.4–0.7 mcg/kg/h IV infusion).

- The nasal mucosa should be anesthetized if a nasal intubation is planned and there are no contraindications. Administer two sprays of oxymetazoline or phenylephrine solution into each nostril. If not contraindicated by mechanism of injury, spray

50 mg of lidocaine into each nostril using a plastic intravenous catheter and syringe.

- Anesthetize the pharynx. Hold the tongue with a gauze pad and apply 50 mg of lidocaine to both palatoglossal arches. This can be done by soaking a gauze pad with lidocaine and placing it against the palatoglossal arch.
- Anesthetize the hypopharynx: Hold the tongue with a gauze pad and apply 100 mg of lidocaine to the back of the tongue using a syringe and a plastic IV catheter. Continue to hold the tongue for several minutes to encourage aspiration of the lidocaine.

Consider a superior laryngeal block: Draw 4mL of lidocaine 2% into a 5-cc syringe with a 25 g needle. Palpate the hyoid bone just cephalad to the thyroid notch. Using the thumb and first finger of one hand, the lateral aspects of the hyoid can be identified bilaterally. Insert the needle just below the most lateral aspect of the hyoid on one side, feeling for resistance of the thyrohyoid membrane. If blood or air can be aspirated, withdraw the needle and reposition. Inject 2 mL of local anesthetic into the membrane. Repeat the block on the contralateral side.

- Anesthetize the trachea: Identify the cricothyroid membrane approximately 1–1.5 cm below the thyroid notch. Insert a needle attached to a syringe filled with 4 mL of lidocaine 2%–4% through the membrane with constant aspiration until air is easily aspirated. After warning the patient that he or she may cough, inject the lidocaine rapidly into the trachea.

Complications

- Failure to provide sufficient airway analgesia may result in coughing, regurgitation, and aspiration.
- Oversedation may cause apnea or airway obstruction.

Special Considerations

Any intubation device, which is used in the asleep patient, may also be used for awake intubation.

- If a FOB is used, lidocaine can be injected through the working channel to augment tracheal analgesia.
- If a video laryngoscope is being used, an atomizer can be used to apply lidocaine to the vocal cords.
- If retrograde intubation is being performed (as described), local anesthetic can be injected during the cricothyroid puncture.

Cricothyroidotomy

Cannula (Needle) Cricothyroidotomy

Indications

- Cannula (needle) Cricothyroidotomy is a method of minimally invasive access into the airway for the purpose of emergent re-oxygenation.
- This procedure should only be performed by personnel who have received proper training and have the proper equipment available because this procedure is associated with high complication and failure rates. Clinicians should pursue advanced airway training prior to using this technique.

Contraindications

- Inability to identify the cricoid-thyroid membrane
- Transected airway
- Laryngeal injury
- Surgical cricothyroidotomy is not recommended in patients <12 years of age unless the cricothyroid membrane is clearly identifiable.

Equipment Checklist for Needle Cricothyroidotomy

- Specialized translaryngeal catheter or 14-gauge IV needle when the patient must be oxygenated but catheters are not available. Standard IV catheters are prone to kinking catheters available from Cook Critical Care, (Bloomington, IN) and VBM (Germany).
- 10-cc syringe
- High pressure (20–50 psi) oxygen source with pressure reducing regulator

Procedure

- Anatomic landmarks: The cricothyroid membrane is one finger-breadth below the thyroid notch. The larynx is immobilized with the left thumb and middle finger. The index finger is then used to identify the thyroid cartilage and the cricothyroid membrane.

Needle Cricothyroidotomy

- Clean and drape the anterior aspect of neck (if time permits).
- Palpate the larynx and identify the cricothyroid membrane as described.
- Stabilize the larynx with the thumb and middle finger.

- Attach the needle-cannula to a 10-mL syringe (with or without fluid).
- Puncture the skin and cricothyroid membrane at a 90-degree angle to the plane of the neck while continuously aspirating until air can be aspirated from the larynx.
- Once air is aspirated, aim the needle-cannula 45 degrees caudad, insert the cannula into the airway, and withdraw the needle.
- Confirm aspiration of tracheal air.
- Attach the regulated high-pressure oxygen source and inject oxygen at 20 cm H₂O pressure for 1 second. Repeat with an inspiration: expiration ratio of 1:3.
- Adjust timing and pressure to achieve chest expansion and recoil.

Large-Bore Cricothyroidotomy (Melker)

Percutaneous Large-Bore Cricothyroidotomy

- Clean and drape the anterior aspect of the neck.
- Immobilize the trachea with the thumb and middle finger and feel the cricothyroid membrane with the index finger.
- Make a vertical incision over the cricothyroid membrane—first through the skin and subcutaneous tissue.
- Attach the included stainless steel needle to a syringe (with or without fluid) and insert it through the cricothyroid membrane into the trachea at a 90-degree angle to the plane of the neck. Aspiration of air confirms that the needle is in the trachea.
- Aim the needle 45 degrees caudad.
- Remove the syringe and pass a guide wire through the needle.
- Ensure that the wire is directed caudad, then remove the needle.
- Pass the assembled dilator-airway (included in the kit) over the wire and into the airway.
- Remove the dilator and inflate the airway cuff (if included).
- Secure the airway to skin and ventilate with a self-inflating bag or the anesthesia machine circuit.

Complications

- Bleeding from the cricothyroid artery or anterior thyroid vein
- Barotrauma or pneumothorax caused by jet ventilation
- Subcutaneous emphysema caused by high-pressure air forced into the subcutaneous space
- Injury to the posterior wall of the trachea

Special Considerations

- In a “can-not-ventilate, can-not-intubate” situation, cricothyroidotomy provides a definitive airway more quickly than a tracheostomy.
- Percutaneous cricothyroidotomy requires prior training and experience.

Double Lumen Endotracheal Tube

Definition

A double lumen endotracheal tube (DLET) can be inserted into either the left or right main stem bronchus in order to selectively ventilate one or both lungs.

Indications

- Selective one-lung ventilation for procedures including lung resection, surgeries of the thoracic esophagus, spine, descending thoracic aorta, video assisted thoracoscopy, and minimally invasive mitral valve surgery via a thoracotomy.
- Selective one-lung ventilation in patients who have bullae or a bronchopleural fistula
- Protection of one lung from the contamination (e.g., blood or infected material) from the opposite lung
- Unilateral lung lavage

Contraindications

- Known or anticipated difficult airway
- Tracheal stenosis or a tracheal stent
- Rapid sequence intubation is a relative contraindication

Equipment Checklist

- Average tube sizes for adults: 35 Fr (for women <160 cm), 37 Fr (for women >160 cm), 39 Fr (for men <170 cm), and 41 Fr (for men >170 cm). Each DLT exists for the left main bronchus and right main bronchus. The left DLT is usually used because of ease of placement due to the variable origin of the right upper lobe.
- Tubes in sizes 28 Fr and 32 Fr are available for pediatric patients.
- Surgical clamp for either the bronchial or tracheal connector.
- Pediatric FOB to verify tube position and to examine the bronchi if necessary
- Lubrication and defogger for the FOB
- DLT suction catheters

Procedure

- Evaluate the airway. If the patient may have a difficult airway, consider using a FOB to intubate with a single lumen ETT, switch to a DLT over a tube exchange catheter.
- If a difficult airway is not anticipated, preoxygenate the patient for 5 minutes prior to induction.
- After preoxygenation, proceed with induction of general anesthesia.
- Direct the tip of the DLT anteriorly, then place it through the vocal cords under direct laryngoscopy.
- After the bronchial cuff is past the vocal cords, remove the DLT stylet.
- Rotate the DLT 90 degrees either to the right or left depending on the bronchus to be intubated and continue to push the tube distally until resistance is encountered (usually 28–30 cm).
- Inflate the tracheal cuff and connect the breathing circuit. Auscultate bilaterally breath sounds and confirm the presence of ETCO_2 .
- Insert FOB into the tracheal lumen and identify the carina. If the bronchial cuff is in the desired bronchus inject 1–2 mL of air into the bronchial cuff and verify that it does not herniate above the carina.
- If the bronchial cuff is not visualized, withdraw the DLT after deflating the tracheal cuff. After the bronchial cuff is observed, inflate the tracheal and bronchial cuffs and again verify the positioning of the bronchial cuff relative to the carina.

Complications

- Hypoxemia during one lung ventilation. Some patients do not tolerate OLV. This could also be caused by a malpositioned DLT.
- Trauma, including vocal cord damage, abrasion, laryngitis, and perforation.
- Inadvertent suturing of the DLT during surgery on a bronchus.
- If the patient develops hypoxemia, confirm proper location of DLT by FOB. Determine if adequate OLV tidal volumes are being delivered 4–6 mL/kg, adjust accordingly, Supply 100% oxygen. If no improvement is observed, provide CPAP to the nonventilated lung. Positive end-expiratory pressure can be added to the ventilated lung. If these maneuvers do improve oxygenation, inform the surgeon that bilateral ventilation is required.

Special Considerations

- A double lumen tube (DLT) is preferred in patients with blood or infectious contamination because it permits suctioning of the isolated lung.
- Verify the position of the DLT after the patient is in the final position to rule out migration of the tube.

Further Reading

Campos JH. Lung isolation techniques for patients with difficult airway. *Curr Opin Anaesthesiol*. 2010; 23: 12–17.

Femoral Vein Catheter

The femoral vein is the preferred route for central venous cannulation in an emergency because the femoral vessels are easier to find and offer direct access to the central circulation. Catheterization of the femoral vein does not have some of the risks associated with internal jugular and subclavian vein catheterization (e.g., arrhythmias, hemothorax, pneumothorax, phrenic nerve, recurrent laryngeal nerve, thoracic duct injury, cardiac tamponade, and valvular injury).

403

Indications

- Inability to obtain peripheral venous access
- Administration of blood, fluids, and medications in an emergency
- Central venous pressure monitoring
- Parenteral nutrition
- Plasmapheresis
- Dialysis
- Insertion of pulmonary artery catheter
- Insertion of a transvenous pacemaker
- Venous access in patients with superior vena cava syndrome

Contraindications

- Adequate peripheral access
- Patient refusal or uncooperative patient
- Deep venous thrombosis or IVC filter
- Abdominal trauma or known IVC trauma
- Burn, infection, or skin damage at insertion site

Equipment Checklist

- Sterile prep solution
- Sterile drapes
- Sterile gown and gloves
- Central venous catheter kit

Technique

- Position the patient supine.
- Prep the site with chlorhexidine alcohol solution (preferred) or povidone iodine solution.
- Put on mask, sterile gown, and sterile gloves. Apply a sterile drape to the site.
- Locate the femoral artery. It is usually 1–2 cm distal to the inguinal ligament and medial to the femoral artery.
- If the patient is awake, inject local anesthetic (lidocaine 1%) subcutaneously, then infiltrate the deeper tissue. Aspirate prior to injecting to avoid inadvertent intravascular injection.
- Insert a needle attached to a 10-cc syringe medial to the femoral artery. After the needle has passed through the skin, apply negative pressure to the syringe while advancing in a cranial direction. Venous blood will fill the syringe when the needle enters the femoral vein.
- Remove the syringe, leaving the needle in the vessel and check for pulsatile (arterial) flow. If a pressure transducer is available, connect it to the needle and verify that a venous pressure and tracing is displayed. In an emergency in which a pressure transducer is not available, aspiration of nonpulsatile, dark blood suggests (but does not confirm) that the needle is in the vein.
- Insert the guide wire through the needle to a depth of 20 cm.
Do not let go of the guide wire.
- After removing the needle over the wire, use the supplied scalpel make an incision along the wire.
- If inserting a triple lumen catheter, slide the supplied dilator over the wire through the skin to the same depth at which the needle was advanced.
- Remove the dilator while leaving the guide wire in place. Advance the catheter over the wire. Slowly remove the wire from the vein, advancing it into the catheter until the end emerges from the distal port. Grasp the end of the wire and slide the triple lumen over the wire and into the femoral vein. Remove the wire and suture the femoral catheter to the skin.
- If inserting a percutaneous sheath, the dilator and catheter are inserted as one unit. Place sheath/dilator assembly over the wire, withdrawing the wire until it emerges from the dilator.

Make a skin incision along the guide wire and slide the sheath and dilator together through the skin. Holding the wire and dilator in one hand, slide the sheath off of the dilator into the femoral vein. Remove the dilator and guide wire as a unit. Secure the catheter to the skin.

Special Considerations

- Femoral vein catheterization is associated with the highest rate of infection and thrombosis.
- The anatomic relationship of the femoral vein and artery can be variable. The risk of arterial puncture and hematoma should be considered, with increased risk in the anticoagulated patient, especially an anticoagulated patient.
- The femoral vein is the preferred route of access during cardiopulmonary resuscitation because it is not necessary to stop chest compressions or discontinue intubation attempts during cannulation.
- Puncturing the femoral vein above the inguinal ligament may cause a retroperitoneal hematoma.
- Ultrasound guided femoral catheterization is less likely to result in an arterial puncture and hematoma than are techniques involving the use of anatomic landmarks.

Complications

- Hematoma
- Inadvertent arterial catheterization
- Intra-abdominal injury, such as bowel perforation
- Retroperitoneal hematoma
- Infection
- Femoral nerve injury
- Air embolism
- Pseudoaneurysm
- Femoral artery or venous thrombosis
- Psoas abscess
- Hip infection

Intubating Laryngeal Masks

The intubating laryngeal masks (ILM) can be used for ventilation and as a tracheal intubation device. Intubating laryngeal masks are similar to the classic laryngeal mask airway (LMA), but include features that facilitate blind or fiberoptic bronchoscope (FOB) placement of a tracheal tube. The barrel of an ILM has a larger diameter than does a typical supraglottic airway, is often more rigid, and is

molded with a fixed or pliable anatomic (right angle) curve. These devices perform well in a can-not-intubate/can-not-ventilate situations.

Indications

- Difficult intubation, difficult ventilation or both
- Elective ventilation or tracheal intubation

Contraindications

- Elective use in the patient at risk for gastric content aspiration

Equipment Checklist

- Intubating laryngeal mask airway (Teleflex Medical, Research Triangle Park, NC; Ambu, Inc., Glen Burnie; Intersurgical, Liverpool, NY; Mercury Medical, Clearwater, FL). Follow manufacture recommendations for size (e.g., for the Teleflex Fastrach: Size 3 is preferred in children older than 12 years of age, size 4 in adults 50–70 kg, and size 5 in adults 70–100 kg.)
- Cuffed endotracheal tube for blind intubation: Use a straight, wire-reinforced, beveled tracheal tube with a Teleflex Fastrach. These are distributed by the manufacturer. Any tracheal tube can be used with devices from other manufacturers.

Procedure

- Intubating laryngeal masks are generally used in anesthetized patients.
- Check the integrity of the cuff and lubricate the ILM with a water-soluble lubricant.
- Position the patient supine with the head and neck in neutral position.
- Hold the device in the left hand while opening the patient's mouth with the right hand.
- Insert the ILM with the posterior cuff tip pressed against the palate and pharyngeal wall. When inserting a Teleflex Fastrach, place in the mouth with the handle parallel to the chest wall. Keep the posterior surface against the palate and pharyngeal wall.
- Continue posterior pressure until resistance occurs and the proximal barrel is parallel with the oral cavity
- Once the device is in place, inflate the cuff and confirm the position with gentle ventilation of the lungs.
- Pass the endotracheal tube through the ILM. If using a Teleflex Fastrach tube, the longitudinal black line should be facing cephalad during insertion. This ensures correct bevel orientation.

- Insert the endotracheal tube 15 cm (to the horizontal black line on Teleflex Fastrach tube) and then advance it another 5 cm.
- If resistance is encountered, gently lift the ILM anteriorly (the Verghese maneuver) while advancing the ETT into the trachea. This maneuver seals the cup of the LMA against the laryngeal orifice.
- Once the ETT is in place, inflate its cuff and confirm its position by ventilating the patient and observing for CO₂ return.
- After an emergency intubation, the ILM can be left in situ while expert advice is sought.

Complications

- The epiglottis may fold as ILM is advanced, occluding the airway. If this occurs, gently withdraw the inflated ILM 6 cm along the access of insertion, and then reintroduce it.
- Do not inflate the ILM for longer than 15 minutes because it can exert excessive pressure on the surrounding tissues.

Special Considerations

- Select the appropriate size ILM to ensure correct placement of the device.
- If the distal end of ILM folds, insert it while it is partially inflated.
- The ILM is a ventilation and an intubation device. If ventilation is possible with the ILM but intubation is difficult, the clinician should continue ventilation until help arrives.

One Lung Ventilation

Absolute indications for one lung ventilation (OLV) include positive pressure ventilation of one lung in the presence of a bronchopleural fistula or bullae in the contralateral lung and the isolation of one lung from the contamination of blood or infected material from the opposite lung. Relative indications for OLV include lung resection, video-assisted thoracoscopy; surgery of the thoracic esophagus, spine, descending thoracic aorta; and minimally invasive mitral valve surgery via thoracotomy. Bronchial blockers and double lumen tubes are the two devices used most commonly to achieve OLV.

Bronchial Blocker: Definition

A bronchial blocker (BB) device is inserted through a single lumen endotracheal tube after tracheal intubation. One-lung ventilation is achieved by inserting the bronchial blocker into the left or right main stem bronchus and inflating the cuff.

Indications

- One lung ventilation
- Known or potential difficult airway
- Difficulty or inability to place a double lumen tube
- Pre-existing ETT in situ
- Patients who are anticipated to require postoperative mechanical ventilation
- One lung ventilation in patients who have a tracheotomy
- Selective lobar isolation

Contraindications

- Patients who are undergoing a lung sleeve resection
- Pathology (e.g., tumor) in the main stem bronchus
- Lack of fiberoptic bronchoscope to confirm placement
- Existing bronchopleural fistula
- Right lung isolation in a patient in whom the takeoff of right upper lobe is above the carina

Equipment Checklist

- Minimum size of the ETT should be 7.5 mm; a larger ETT is preferable.
- Pediatric fiberoptic bronchoscope
- Lubricant for fiberoptic bronchoscope and bronchial blocker
- Defogger for fiberoptic bronchoscope
- Bronchial blocker device

Preparation

- Evaluate the airway to determine if ventilation or intubation may be difficult.
- Discuss the use of a bronchial blocker with the surgical team.
- Confirm that the ETT can accommodate both the bronchial blocker and FOB prior to intubation by placing both into the ETT

Technique

- Check the bronchial cuff by placing the specified amount of air from the manufacturer. Once cuff integrity is confirmed, remove the air from the cuff.
- Lubricate both the bronchial blocker and fiberoptic bronchoscope.
- Intubate the patient with an ETT with an outer diameter of at least 7.5 mm.
- Insert the FOB into the ETT and identify the region to be isolated.

- Insert the bronchial blocker into the ETT and advance it until visualized with the fiberoptic bronchoscope.
- Direct the bronchial blocker into the bronchus or lobe that is to be blocked.
- Inflate the cuff with the specified volume of air and confirm that the bronchial blocker has not moved.
- Remove the fiberoptic bronchoscope and open the bronchial blocker lumen to permit the lung to deflate.

Complications

- Trauma to airway, including bleeding, rupture, pneumothorax, or bronchopleural fistula.
- Overinflation of the cuff can reduce mucosal blood flow and cause ischemia.
- Airway occlusion can occur if the bronchial blocker cuff migrates into the carina.
- Hypoxemia may occur in a patient who is unable to tolerate one-lung ventilation.

Special Considerations

- Bronchial blockers can easily become dislodged. Verify that the bronchial blocker is in the correct location if the patient is repositioned.
- Unlike double lumen tubes, bronchial blockers cannot be suctioned.
- The lumen of the bronchial blocker may become occluded during surgery. If this happens, it can usually be reopened by irrigating with 2–3 cc of normal saline.

Further Reading

Campos JH. Lung isolation techniques for patients with difficult airway. *Curr Opin Anaesthesiol.* 2010; 23: 12–17.

Campos JH. Update on selective lobar blockade during pulmonary resections. *Curr Opin Anaesthesiol.* 2009; 22(1): 18–22.

Retrograde Intubation

Definition

An intubation technique in which a guide wire is inserted through the cricothyroid membrane and retrieved from the mouth (or nose). A tracheal tube is then threaded over the guide wire into the trachea.

Indications

- Retrograde intubation is indicated when conventional techniques for intubation either fail or are not feasible.
- Unstable cervical spine
- Excessive secretions or blood in the airway
- Oropharyngeal malignancy
- Small mouth opening (direct laryngoscopy and fiberoptic intubation requires that the mouth open 3–4 cm)
- Failed intubation by other means in an otherwise stable patient
- Elective intubation of a patient with a known or suspected difficult airway

Contraindications

- Inability to access the cricothyroid membrane or local infection
- Coagulopathy
- Tumor or trauma involving the larynx
- Retrograde intubation can be time-consuming and should be considered to be an elective procedure.

Equipment Checklist

- Intravenous catheter or Tuohy needle: The size should be able to accommodate the following wire.
- Wire: 0.035-inch guide wire (75 cm long; 2–2.5 times the length of a standard endotracheal tube)
- 5- to 10-mL syringe
- Magill forceps
- Endotracheal tube
- Complete kits are available (e.g., Cook Critical Care). Using a kit ensures that all necessary equipment is available.

Procedure

- Position the patient supine with the neck slightly extended.
- If the patient is conscious, prepare as suggested in the section on awake intubation (see page 396).
- Clean and drape the anterior part of the neck.
- Identify the cricothyroid membrane and, if the patient is conscious, infiltrate the skin and subcutaneous tissue over the cricothyroid membrane with 1% lidocaine.
- Insert the intravenous catheter into the trachea through the cricothyroid membrane. Confirm its position by aspirating air.
- Direct the catheter cephalad. The 0.035-inch guide wire is threaded through the catheter and retrieved from the mouth.

A laryngoscope and Magill forceps may be required to reach the proximal end of the guide wire.

- Remove the catheter and place a small clamp over the distal end of the guide wire to prevent it from slipping out.
- Thread the ETT over the proximal end of the guide wire. Maintain tension by gently pulling on both ends of the wire while the ETT is being passed.
- Remove the guide wire from the cricothyroid membrane and pass the ETT further down into the trachea.

Complications

- Subcutaneous emphysema
- Wire travels in a caudad direction, causing coughing
- Failure to achieve tracheal insertion of the wire
- Inability to pass the larger endotracheal tube past the entrance of the larynx.

Special Considerations

- The guide wire can be threaded through the Murphy's eye of the endotracheal tube to provide better control over the distal end of the endotracheal tube.
- After the ETT enters the trachea, a soft bougie can be passed before advancing the tube further.
- If resistance is encountered when passing the ETT, the tube is most likely impinging on the pyriform fossa. Gently rotate the tube while advancing it.

Transcutaneous Pacing

Definition

Cardiac pacing through external cardiac defibrillator pads. Transcutaneous pacing is most commonly used in patients with cardiac rhythm that is causing hemodynamic instability.

Indications

- Unstable and symptomatic bradycardia
- Mobitz type II second-degree AV block
- Third-degree heart block
- Exchange or repair of an existing permanent pacemaker
- During re-do cardiac surgery
- Overdrive pacing in patients with atrial flutter and monomorphic ventricular tachycardia

Contraindication

- Asymptomatic patients with stable rhythm
- Hypothermia-induced bradyarrhythmias

Complications

- Incorrect pacing mode causing tachycardia or atrial or ventricular fibrillation
- Pain
- Skin burn
- Diaphragm irritation

Equipment Checklist

- Electrocardiogram
- External pacemaker
- Defibrillator
- Pacing pads

Pad Application

- If the patient has thick hair, shaving may be required prior to pad application.
- Place the anterior pad at the cardiac apex.
- Place the posterior pad medial to the right scapula.

Transcutaneous Pacemaker Operation

- In the conscious patient: Apply the pads as above and connect them to the external pacemaker. Set the current to 0 mA and the heart rate to 80 bpm in the demand (synchronous) mode. (When set to *synchronous mode*, the pacer only delivers a pulse when no cardiac complex is sensed in a set amount of time.)
- Increase the output in 10 mA increments until capture is achieved as indicated by a QRS complex after each pacing spike on the ECG. Capture is confirmed by feeling a pulse consistent with the pacing rate.
- The pacer should be set to deliver 10 mA above the threshold current.
- In an unconscious patient, set the pacer to the maximum output (200 mA). Decrease the output in 10-mA increments until capture is lost. The final current delivery should be set to 10 mA above threshold.
- If the pacer does not capture in the synchronous mode, select the asynchronous mode, in which the pacer delivers pulses at at preset time intervals without sensing underlying intrinsic cardiac activity.

Special Considerations

- Transcutaneous pacing is not recommended for treatment of asystole. Multiple randomized controlled trials showed no improvement in rate of admission to hospital or survival to hospital discharge when transcutaneous pacing was used in the prehospital setting or in the emergency department.
- It has been reported that a prognosis is poorer when transcutaneous pacing is used for asystole as compared to bradycardia.

Further Reading

Epstein AE, DiMarco JP, Ellenbogen KA, et al. ACC/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the ACC/AHA/NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices): developed in collaboration with the American Association for Thoracic Surgery and Society of Thoracic Surgeons. *Circulation*. 2008; 117(21): e350–408.

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Ryan TJ, Anderson JL, Antman EM, Braniff BA, Brooks NH, Califf RM, Hillis LD, Hiratzka LF, Rapaport E, Riegel BJ, Russell RO, Smith EE III, Weaver WD. ACC/AHA guidelines for the management of patients with acute myocardial infarction: executive summary. *Circulation*. 1996; 94: 2341–2350.

Transport of a Critically Ill Patient

The intrahospital transport of a critically ill patient carries a significant risk for life-threatening adverse events, including:

- Hypoxia
- Severe hypotension or hypertension
- Arrhythmias
- Cardiac arrest
- Agitation
- Intracranial hypertension
- Inadvertent extubation
- Bronchospasm
- Pneumothorax
- Medication errors
- Hemorrhage
- Equipment failure

Objectives of safe transport include:

- Stabilization of the patient prior to transport
- Appropriate level of monitoring (e.g., vital signs, pressure tracings, intracranial pressure)
- Maintaining consistent ventilator settings prior to and throughout patient movement
- Communication between the care teams at the points of departure and destination to decrease transport time and minimize the time that the patient away from a critical care environment
- Provision of sedation to prevent the patient from harming himself or herself by removing ETTs, IVs, chest tubes, drains, or pacer wires
- Anticipation of and preparation for potential deterioration of the patient's condition

Transport team members must be qualified to provide the same level of care as in the ICU, and must be prepared to correct any adverse events. Medications, fluids, a self-inflating bag and mask, airway management supplies, and resuscitation equipment must be immediately available throughout transport.

Equipment Check

- Transport monitor with capabilities to measure noninvasive blood pressure, arterial and venous waveforms, ECG, and pulse oximetry
- Transport ventilator with a full oxygen tank
- Self-inflating bag and mask
- Airway management equipment; ETTs and stylets, oral airway, laryngoscope and blades, and empty 10-cc syringe
- Emergency medications for hemodynamic support, sedation, and intubation
- Intravenous fluids and infusion pumps (if need is anticipated)
- All electronic equipment, including monitors, infusion pumps, and respiratory equipment, must have sufficient charge for the duration of the transport.

Ready for Transport

- Begin patient handoff when all required personnel for transport are present.
 - Airway: Confirm that the ETT is secure, suction if necessary, and verify that airway management equipment is ready.
 - Anesthesia: Continue patient sedation during transport. Anticipate the need to increase the level of sedation during

transport. If the patient requires neuromuscular blocking agents, these should be administered prior to leaving the room.

- **Breathing:** If possible, a respiratory therapist should accompany the patient. Bring a portable ventilator (preset to the patient's ventilator settings) and a full oxygen tank.
- **Bed:** The handoff discussion should include specific patient positioning requirements (e.g., elevated head for intracranial hypertension or flat position for a patient with an unstable cervical spine).
- **Circulation:** Confirm that monitoring equipment is functioning properly. Stabilize the blood pressure and heart rate before moving the patient. Bring medications, IV fluids, and check infusion bag volumes to verify sufficient volume for the expected transport duration.
- **Destination:** All the team members must know the destination. Confirm that the personnel at the new location are ready to receive the patient.

Transfer of Patient Care in the Intensive Care Unit

415

- Place patient on ICU monitors and record the vital signs for the transfer note.
- Connect patient to the ICU ventilator. Use settings that worked well in the operating room.
- Administer appropriate medication and fluids, and adjust ventilator settings to stabilize the patient's vital signs.
- If the patient is receiving medication infusions (e.g., sedatives, vasoactive drugs), plug the pumps into wall sockets to prevent battery failure.
- Sign out the patient to the ICU team. Include pertinent intraoperative events (including any adverse events), fluid status, medication administration, including timing of last doses, issues related to airway management, and intraoperative laboratory values.

Further Reading

Fanara B, Manzon C, Barbot O, Desmettre T, Capellier G. Recommendation for the intra-hospital transport of critically ill patients. *Crit Care*. 2010, 14: R87.

Transvenous Pacing

Definition

Insertion of pacing electrodes through a vein into the right atrium and/or ventricle for treatment of severe symptomatic bradycardia.

Indications

- Symptomatic bradycardia
- Mobitz type II second-degree AV block
- Third-degree heart block
- Exchange or repair of an existing permanent pacemaker
- Overdrive pacing in patients with atrial flutter and monomorphic ventricular tachycardia
- Failure to capture with transcutaneous pacing
- Temporary electrode insertion after transarterial valve replacement surgery (high incidence of bradycardia and heart block)

Contraindications

- Hypothermia induced bradyarrhythmias
- Asymptomatic patients who have a stable cardiac rhythm

Equipment

- Physiologic monitors, including ECG, blood pressure, and pulse oximetry
- Advanced cardiac life support medications and supplies
- Airway management equipment
- Defibrillator with transcutaneous pads
- Sterile prep solution, sterile gloves and gown, face mask, and head cover
- Local anesthetic (e.g., lidocaine 1% solution)
- Pacer percutaneous sheath introducer kit
- Pacing leads
- Suture material
- Fluoroscopy for lead placement location if available
- External pacer generator

Technique

- If the patient is conscious, obtain informed consent.
- Place ECG leads on the patient
- Refer to *Ultrasound Guided Central Venous Access* (page 418) to obtain central venous access for introducer sheath. (The most common venous access sites are the internal jugular or subclavian vein.)

- Insert a balloon tipped pacing wire through the introducer. When the tip is in the vein inflate the balloon and connect to the V1 lead of the ECG.
- Advance the pacing wire while observing the ECG. The waveform will initially display a small P and a larger QRS.
- As the tip of the wire is advanced into the RA the P wave will be larger than the QRS.
- When the catheter enters the RV, the P wave decreases in amplitude and the QRS becomes larger. When the tip contacts the RV wall, the V wave amplitude will increase and ST segment elevation may be noted (*injury current*), indicating proper contact for pacer conduction.
- Connect the lead to the external generator. If perfusion is compromised, use the highest available current output then decrease slowly until pacing capture is lost. Increase the current to regain capture.
- If the patient is hemodynamically stable, set the pacer rate to 20 beats above the underlying intrinsic heart rate with a low output. Increase current incrementally until pacer capture is obtained. Current setting should be 2–3× capture threshold.
- After insertion, obtain a chest X-ray to confirm placement and rule out pneumothorax.

Complications

- Failure to detect arrhythmias
- Induction of atrial and ventricular fibrillation and tachycardia
- Hematoma
- Pneumothorax
- Arterial puncture
- SVC, RA, RV perforation leading to pericardial tamponade
- Infection
- Venous air embolism

Special Considerations

- Transesophageal echocardiography can be used to verify lead position during insertion.
- Flexible catheters are preferred because they carry less risk of perforation than do stiff catheters.
- Loss of capture can occur if the lead is displaced. If this happens, increase output to determine whether capture can be regained. Request a chest X-ray to verify lead position.
- Acidosis, electrolyte abnormalities, hypoxia, and antiarrhythmic medications can increase the capture threshold and cause loss of pacing.

Further Reading

Epstein AE, DiMarco JP, Ellenbogen KA, et al. ACC/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (writing committee to revise the ACC/AHA/NASPE 2002 guideline update for implantation of cardiac pacemakers and antiarrhythmia devices): developed in collaboration with the American Association for Thoracic Surgery and Society of Thoracic Surgeons. *Circulation*. 2008; 117(21): e350–408.

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Ultrasound-Guided Central Venous Access

418

Definition

Use of ultrasonographic imaging to facilitate insertion of a catheter into the external jugular, internal jugular, femoral, or subclavian veins. Ultrasonic guidance also can be used to gain access to central arteries (e.g., the femoral artery), peripheral arteries (e.g., the radial artery) and peripheral veins.

Contraindications

- Inexperience with ultrasound use
- Inadequate knowledge of anatomic landmarks and ultrasound structures

Anatomy

- Arteries appear pulsatile on two-dimensional ultrasound, have a characteristic appearance on color flow Doppler, and cannot be compressed when mild to moderate pressure is applied with the probe.
- Veins do not appear nonpulsatile on two-dimensional ultrasound (except for the internal jugular veins in patients with severe tricuspid regurgitation), can be compressed when mild pressure is applied with the probe, and distend when there is increased venous pressure (e.g., Trendelenburg position when examining the jugular or subclavian veins and during tourniquet application when examining peripheral veins).

Equipment Checklist

- Ultrasound machine and probes:

Linear transducer probes are useful for smaller sectors and high resolution. Curved transducer probes are useful for scanning a wide field

- Central line kit, including sterile prep solution and drapes
- Sterile sheath and ultrasound gel to cover the ultrasound probe
- Sterile gloves, sterile gown, face mask, and head cover for operator
- To access a peripheral artery or vein, all of the above supplies except the central line kit. Supplies for peripheral IV or arterial line catheter should be available.

Technique for Right Internal Jugular Central Line Placement

- Turn the patient's head to the left <30 degrees if possible.
- Apply ultrasound gel to the probe and orient to medial and lateral on the probe to the image on the ultrasound monitor.
- Scan the neck with the ultrasound probe after gel has been applied to identify the carotid and internal jugular veins. The carotid artery in most patients will be located medial to the internal jugular vein.
- Place the patient into the Trendelenburg position, with the head down 15–25 degrees. (The patient should be secured to the bed or operating room table.)
- Don a face mask, hat, sterile gloves and gown using standard aseptic technique.
- Open central line kit using sterile technique.
- Scrub the neck with sterile prep solution and apply a sterile drape.
- Open the sterile ultrasound sheath and place gel inside. An assistant should place the probe inside the sheath, leaving the external part sterile.
- Pull the sheath tight to eliminate air bubbles between the probe and the sheath.
- Prepare the central line kit.
- Palpate the carotid pulse and apply ultrasound gel to the skin.
- Orient the ultrasound probe in the transverse (perpendicular to flow) direction on the neck and identify the carotid and internal jugular vein. Scan the neck caudal and cephalad to find the area where the carotid and internal jugular are separated (or overlap the least), and the internal jugular is largest in diameter.
- If the patient is awake, infiltrate the skin with 1–2 mL of lidocaine 1%. If the patient is anesthetized, local anesthesia is not necessary.

- Align the ultrasound probe so that the carotid artery is medial and the internal jugular vein lateral. The internal jugular vein should be aligned with the middle of the probe.
- Attach a needle or catheter large enough to accommodate a wire to a 5-mL syringe. Place the needle at a 45-degree angle in the middle of the transducer probe and press down (without piercing the skin). It should be possible to see an indentation that is aligned with the internal jugular vein.
- Enter the skin while observing that the trajectory is still aligned with the internal jugular vein. After the needle pierces the skin, apply negative pressure to the syringe. If the needle is seen to approach the artery, pull it back to the skin and direct it in a more lateral direction.
- As the needle tip approaches the vein, an indentation should be seen on the monitor. Direct eyesight toward the syringe as it is advanced to watch for aspiration of venous blood.
- Remove the syringe and thread the central line guide wire to a depth of 20 cm. The ultrasound probe should confirm the wire is in the internal jugular vein by both transverse and longitudinal imaging.
- Remove the needle, make a skin incision in the direction of the wire, and advance the dilator to the same depth as the needle was inserted.
- Advance the central line over the wire and secure using suture material.
- Confirm the position with a chest x-ray.

Complications

Special Considerations

- The frequencies used most commonly for catheter insertion are 2–12 MHz.
- Higher frequencies offer better resolution with decreased depth of penetration.
- Lower frequencies offer increased depth of penetration with poorer resolution.

Further Reading

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Chapter 16

Disaster Management

Michael J. Murray

Pre-Disaster Planning 423
Response to a Disaster 425

Oklahoma City; 9/11; the tornado that struck Joplin, Missouri; hurricanes Sandy, Ike, and Katrina; and the Boston Marathon bombing have left indelible marks on our national consciousness. All of us have either been directly affected by or witnesses to the tragedies that occurred. We can't predict when or where the next major disaster such as an earthquake in California or Haiti will occur (It is far more likely to be from a natural cause than an act of terrorism), but it is possible to prepare and plan. Anesthesiologists should know their departments' and facilities' disaster response plan and what their role will be should a disaster that generates mass casualties occur.

Disaster management in the traditional sense includes disaster prevention, preparedness, relief, and recovery phases. Disasters cannot be prevented, but the scope of the disaster can be mitigated through *disaster prevention* strategies that decrease mortality and morbidity rates by creating effective evacuation plans and by environmental planning (e.g., construction of earthquake-resistant buildings, flood control projects). *Disaster preparedness* is the primary means of minimizing the loss of life and damage, for example, by relocating people and property from an at-risk location, and by providing rescue, treatment, and rehabilitation in a timely and effective fashion. *Disaster relief* activities include relocation and rescue of affected individuals, along with the provision of water, food, and shelter, prevention of communicable disease, restoration of vital services such as electricity and telecommunications, and the provision of emergency health care. *Disaster recovery* includes the reconstruction of infrastructure that might have been damaged or destroyed and re-establishment of routine health care delivery and rehabilitation services. For anesthesiologists' purposes this chapter's emphasis is on pre-disaster preparedness, care of patients who have been injured by the catastrophic event, and mitigation of the effects of disasters on ourselves and on our facilities.

The term *mass casualty* refers to a large number of injuries or deaths that have the potential to overwhelm a hospital or a city's hospital system. The same event that created the increased number of patients might also affect the functionality of the hospital itself. For example, there might be structural damage to a hospital after an earthquake or tornado or interruption of the hospital's electrical power by flood waters. A mass casualty event is a unique process, one that many of us will not personally experience in our lifetimes, but one for which we must be prepared. When these events occur, the best outcomes are achieved with a multidisciplinary team approach.

The terms *mass casualty incident* refers to a situation in which the health care facility has the resources to manage the number of casualties that arrive at the facility. In contrast, a *mass casualty event* occurs when the number of patients overwhelms the hospital, which

does not have the requisite resources to cope with, or manage, the number of patients arriving at the facility. Although a mass casualty event could occur in a rural setting most often they occur in urban centers that have protocols that direct emergency medical technicians (EMTs) and paramedics to transport patients to specific locations based on their severity of illness. After the Boston Marathon bombing, patients arrived at 29 different hospitals, with the most severely injured distributed equally to the several Level I trauma centers in the city. Patients do not, however, always wait for the EMTs or paramedics for evaluation and transportation; this was the case in Tokyo after the subway Sarin nerve gas attacks in 1995 and in Israel during the 1990s after multiple suicide bombings. Patients either transport themselves, or are transported by passersby, to the nearest hospital. Several thousand patients overwhelmed St. Luke's hospital in Tokyo after sarin gas was released by the Aum Shinrikyo terrorist group in 1995. There was no decontamination or triage of the victims. In fact, 20 physicians were exposed to sufficient agent to become casualties themselves, consuming resources and decreasing the number of personnel on the health care team. The distinction between a mass casualty incident and an event is therefore arbitrary and is of not much significance to an anesthesiologist who is called to the hospital because of a disaster.

Pre-Disaster Planning

One limitation that a hospital could have in responding to a disaster is a lack of adequate staffing to manage the number of casualties arriving at the institution. Physicians are notoriously ill-prepared for responding to disasters and must therefore make arrangements for their family, pets, and valuable belongings before attending to professional responsibilities. During Hurricane Katrina, for example, one-third of the hospital's work force would not or could not report to the hospital.

Family Disaster Plan

The four components of a family plan (Box 16.1) are: 1) identification of most likely disaster to occur in geographic area, 2) family disaster plan, 3) checklists, and 4) periodic drills to test the efficacy of the plan.

Each family should create two pre-arranged meeting points. One should be outside the home in case of a fire in the home and the other outside the neighborhood in case of a flood or other widespread disaster. Copies of important documents should be stored in a safe place off-site (e.g., in a bank safety deposit box). All members of the family should know how to

Box 16.1 Family Emergency Preparedness Plan

- Three-day supply
 - Water
 - Food
 - Prescription drugs
 - Cash
- Flashlight
- Battery-powered radio

turn off water and power to the house in case evacuation is required. Provisions for pets or family members who are disabled are critical components of an emergency plan but are often overlooked.

Personal Emergency Preparedness Plan

In addition to a family plan, preparations should be made for responding to a disaster. During an evacuation (e.g., before a hurricane), roads may be so jammed with traffic that they may be impassable, or bridges may be closed by wind or destroyed by an earthquake. It is therefore important to have contingency plans for timely arrival at the hospital during a mass casualty event. As important as having a 3-day supply of food at home, one should have a bag prepared in advance with cash, personal hygiene items and clothes, and a vehicle filled with fuel (ATMs and fuel pumps do not work without electricity). Communication networks are frequently inoperative following a disaster because the circuits are overloaded or because transmission sites and lines were destroyed. In such circumstances a battery-powered radio and/or a satellite telephone might be helpful.

Professional Emergency Preparedness

Maintaining competency in the management of patients injured by a weapon of mass destruction or infected with a biologic agent during an outbreak is difficult if not impossible, so several health care organizations have developed “just-in-time” training tools. A professional emergency preparedness plan should include familiarization with how to access these tools, which offer concise information specific to the patient population being managed at the point of care. The “training” can be downloaded from the Internet, which in the majority of events would be functioning and accessible, but the truly prepared physician would have previously downloaded and stored the information for use on her or his personal digital assistant when needed.

Although hospitals are required to test their facilities' emergency response plan biannually, <15% of anesthesiologists in the United States have ever participated in such drills. Physicians who have participated in mass casualty situations state afterward that being better educated and trained would have helped them to provide better care for patients. If it is not possible to participate in a hospital-wide disaster response drill, an anesthesiologist should at a minimum familiarize himself or herself with the anesthesiology department's plan.

Perhaps of more concern to anesthesiologists responding to a mass casualty incident or event caused by terrorism is that many hospitals do not have the resources to store and maintain equipment that will only be used in the event of a disaster. In a survey conducted by the Centers for Disease Control and Prevention, only 9% of the 789 hospitals that responded were fully prepared for a disaster, and each had on average only 14 personal protective suits. Personnel may also have to arrange for access to an N-95 (face mask) respirator that is capable of capturing 95% of airborne particles between 1 μm to over 100 μm in size over a range of airflows of between 10 L/min to 100 L/min. A retrospective analysis of 47 nurses managing patients with SARS (severe acute respiratory syndrome) in the intensive care unit (ICU) found that wearing an N-95 respirator while the patients were being intubated prevented the nurses from acquiring SARS. It would seem prudent then to have been fitted in advance for an N-95 mask, and to know where that mask was stored.

Disasters not only disrupt the physical environment (e.g., houses, buildings, roads), but, as observed during Hurricane Katrina, they also change social behavior. Hospital emergency response plans should therefore include locking the facility down to control access. One should know how to access the hospital in advance and how to find the command and control center, which is typically in the emergency department.

Anesthesiologists are able to rapidly assess patients with traumatic injury, manage airways, and obtain vascular access. They can also provide sedation, pain control, and comfort care and also have an in-depth knowledge of the drugs used in the treatment of injuries. Therefore, they could be assigned by the control center to assist with triage outside the hospital, to the emergency department, to the operating suite, or to the ICU.

Response to a Disaster

Most people are concerned about a mass casualty event that is caused by the use of a weapon of mass destruction, but >90% of

Table 16.1 Disasters That May Result in Mass Casualties

Natural	Unintentional	Intentional
Weather	Airplane, train, or bus crash	Chemical
Hurricane	Boat sinking	Biologic
Tornado	Fire	Radiologic
Flood	Nuclear power plant accident	Nuclear
Earthquake	Building collapse	Explosion
Infectious	Sports stadium disaster	
Influenza		
Hemorrhagic fever		

the disasters in the world are natural events (Table 16.1). These events, which affect >200 million people annually, are broadly classified as meteorologic, geophysical, and biologic. They are in the national conscience because they occur so frequently, and they are events with which most people are familiar. Physicians likewise, have experience treating the types of injuries and illnesses caused by natural events; patients receive blunt or penetrating injuries, for example, or they develop pneumonitis from an infectious agent. Everyone has heard about the threats posed by weapons of mass destruction (WMD)—chemical, biologic, radiologic, nuclear, and (high-energy) explosive (CBRNE) devices—but few physicians have ever seen or managed a patient injured by WMD. Although these weapons are rarely used, all physicians should understand some of the basic tenets of managing patients injured by WMD.

Natural Disasters

The United States is relatively fortunate because the effects of natural disasters have not been as severe as seen elsewhere in the world, where millions of people are affected each year. Those who have experienced first hand the effects and sequelae of hurricanes, tornadoes, earthquakes, or floods, or who have worked in an ICU providing care to patients with SARS, avian flu, or Ebola know well the impact that these events have had on their communities and themselves.

Meteorological Disasters

In August 2005, hurricane Katrina struck New Orleans, Louisiana. Hurricane Ike struck Galveston, Texas, in September 2008, and in October 2012 hurricane Sandy came ashore just northeast of Atlantic City, New Jersey. Hurricane Sandy was barely a Category 2 hurricane (sustained winds of 96–110 mph) when it made landfall but because it struck during high tide, however, its

storm surge caused significant flooding in New Jersey, New York, and other states along the Eastern seaboard. All three hurricanes flooded hospitals, and many were incapacitated because electrical power was lost or functioning at partial capacity because employees were unable to commute to the hospital. Mass transit ceased, roads were flooded, and the subway system was closed in New York. The United States was fortunate because there were far fewer casualties than those experienced by low-income countries. The economic impact, however, was staggering; the economic damage from hurricane Sandy alone was approximately \$65 billion. Moreover, people still develop common ailments during such disasters. Individuals who develop appendicitis still require treatment, women in labor may require a cesarean section, and patients with coronary artery disease may have an acute myocardial infarction either *de novo* or caused by stress precipitated by the storm. Patients often somehow make their way to the closest health care facility, but if that facility is closed and is in the process of transferring its patients to other hospitals, the newly arriving patients will become part of that exodus. The receiving hospital is then faced with an influx of patients, some of whom are stable and some of whom require emergency intervention. Anesthesiologists practicing at the receiving hospitals or at outlying hospitals, must implement their family and personal emergency response plans, anticipate arrival at the hospital before the hurricane makes landfall, and have the ability to remain self-sufficient for as long as 48–72 hours.

The tornado that struck Joplin, Missouri in 2011 destroyed much of the town, including one of its two hospitals. Paramedics and EMTs worked out of their ambulances to render aid. On May 20, 2013, an EF5 tornado, with peak winds estimated at 210 mph struck Moore, OK, and adjacent areas, killing 24 people and injuring 377 others. Those 377 individuals sought aid wherever possible, and many who could not find a hospital asked for help in pharmacies, and free-standing surgery centers. Whatever the circumstance, anesthesiologists must be prepared to render aid.

Geological Disasters

Earthquakes are not as common as weather-related disasters but still claim more than 1 million victims a year. Because of a lack of building codes, low-income countries tend to have the highest morbidity and mortality. Even developed countries such as the United States can experience significant damage and loss of life, as happened in Northridge, California in 1994. Coastal regions of Indonesia and Japan experienced significant destruction from tsunamis that were caused by earthquakes. Casualties and patients requiring emergency treatment for non-disaster-related problems

(e.g., appendicitis) might need to be transported out of the earthquake zone of hospitals are destroyed or rendered unsafe because of structural damage. Crush injuries and amputations are the most commonly seen injuries in patients who have sustained trauma. Anesthesiologists may be asked to assist at the site of a collapsed building to care for a patient who needs a surgical amputation in order to be extracted from a collapsed building. The aim in such a situation is to provide an anesthetic that is quick and effective (e.g., intramuscular or intravenous ketamine and midazolam) while recognizing that the usual standard of care may be impossible to achieve.

Biological Disasters

Diseases such as SARS and influenza are highly contagious and are as likely to disrupt a community as would be the dissemination of anthrax or smallpox by a terrorist group. The Spanish influenza virus infected approximately 500 million people during the pandemic of 1918–1919, and of those between 50 and 100 million died. Many of those who died were young adults 20–30 years of age, and they died within days of becoming ill. The H₁N₁ influenza epidemic in 2009 also resulted in considerable morbidity and mortality and placed enormous demands on many hospitals. More recently, the outbreak of Ebola Viral Disease (EVD) in West Africa has attracted worldwide interest, and though there were few cases outside of Africa, hospitals and healthcare personnel in high income countries spent considerable time and energy preparing for the worst.

The most critical lesson to be learned from these epidemics is that infected patients or those considered to be infected must be isolated from contact with others in order to achieve the best outcomes. Moreover, health care workers must take the necessary steps to protect themselves. The experience with SARS in Canada emphasizes this point. When the first patients with SARS arrived in Toronto, no one appreciated the etiology of their illness and other patients and health workers contracted the disease. The spread stopped when the scope of the problem was recognized, patients were isolated, and health care workers began to use protective equipment. Because of the experience in Toronto, hospitals in Vancouver were on the alert. Patients with a respiratory illness recently arrived from Asia, were isolated, and the spread of SARS in Vancouver was much less than was observed in Toronto. Implementing strict infection control techniques can also stop the spread of EVD, which is very contagious and associated with a high mortality rate. Because of the social norms in western Africa, however, the person-to-person spread has been significant.

Anesthesiologists may be called upon to use their skills in airway and ventilator management and may also be involved in the

management of patients who are candidates for extracorporeal membrane oxygenation. Providing care to such patients is problematic, however, when wearing personal protective equipment. The suits are not ventilated and are therefore uncomfortable to wear. Providers are unable to work for >60–90 minutes at a time because of heat accumulation, at which point they must decontaminate themselves, disrobe, and rehydrate.

Unintentional Disasters

Crashes

Most crashes, whether of an airplane, bus, or train, do not create a mass casualty event but may create a mass casualty incident. Paramedics and EMTs transported patients to several hospitals, all of which experienced a mass casualty incident, after the Asiana Flight 214 accident in San Francisco in 2013. All of the hospitals that received casualties were, however, able to provide care in a timely fashion. Later that year, after 72 tank cars filled with crude oil derailed and caused a firestorm in Lac-Mégantic, Québec, Canada, the sole local hospital responded appropriately. Recognizing that there were insufficient personnel to handle a mass casualty incident, calls were made to nearby hospitals to request additional medical personnel. Despite the fact that one-half of the downtown was destroyed, 42 people were killed, and five were never accounted for, every casualty received appropriate care. In each of these cases, the emergency response system and the affected hospitals responded appropriately.

Fires

Appropriate management of the airway is one of the most critical components after an industrial accident or fire. Health care facilities near large industrial complexes must be prepared to manage patients with thermal injuries from chemical explosions and must therefore anticipate or know the types of chemicals to which patients may have been exposed.

One hundred people died immediately in a fire that struck a Rhode Island nightclub on February 20, 2003; 200 more were injured and required treatment at the nearby small community hospital. No neuromuscular blocking agents were used to intubate patients who required mechanical ventilation for surgery or to treat pulmonary insufficiency caused by smoke inhalation; the anesthesiologists realized only afterward this was the correct approach, as several patients had glottic edema that made intubation of the trachea difficult.

Patients with thermal injury require intravenous access as soon as possible for administration of intravascular fluids. Multiple protocols exist for the management of patients with thermal injury, but in essence Ringers Lactate solution should be administered

during the first 24 hours at a rate of 1–2 mL/kg/percent body surface area burn. Avoid giving boluses if possible, adjusting the rate of administration of fluids to the urine output. Decrease or increase the Ringers Lactate infusion 20% per hour to maintain a urine output of 30–50 mL/h. Over-resuscitation is as harmful as under-resuscitation; during the 24 hours management of fluids on an hourly basis is critically important.

In patients with soft tissue and skeletal muscle damage, very high creatinine phosphokinase levels correlate with the degree of myoglobinemia. Alkalinizing the urine, along with intravascular volume resuscitation and promotion of diuresis, may protect the kidneys from injury.

Industrial Disasters

Health care workers, especially those who live or work near large industrial complexes, must be prepared to deal with industrial accidents. The Bhopal, India gas tragedy in December, 1984, was one of the worst industrial accidents in recent memory. Methyl isocyanate gas was released into the atmosphere from a tank, killing approximately 3800 people and injuring several thousand more, many of whom required assistance with oxygenation and ventilation. The aftereffects are felt to this day.

The community in which one lives influences the kinds of natural disasters for which planning and preparation are necessary (e.g., a hurricane, a tornado, or an earthquake) and also influences the types of industrial accidents for which one should be prepared. For example, personnel who work at a hospital that is close to a chemical plant should be prepared to provide care for the accidents that might occur at that plant. Although the number of affected people may be fewer than in Bhopal, anesthesiologists might be called upon to render assistance to plant workers who have been injured by toxic fumes.

It is not always possible to anticipate the kinds of casualties that might arise from an industrial accident if there are main line railroad tracks, petrochemical plants or nuclear power plants in the vicinity. However, following an incident, emergency response personnel should be queried for any information about the chemicals to which patients might have been exposed. Railroad tank cars are required to have information about their contents clearly displayed. Most first responders also carry Geiger counters and should be able to provide some information about the need for decontamination of patients arriving from a nuclear power plant accident. On April 17, 2013, only 2 days after the Boston Marathon bombing, an explosion occurred at a fertilizer storage and distribution facility in a small town close to Waco, TX, while emergency personnel were responding to a fire at the

facility. At least 15 people were killed and more than 160 were injured. On June 14, 2013, a chemical-plant explosion killed two people and injured more than 100 at the Williams Olefins petrochemical plant in Geismar, LA. Another explosion the same day killed one and injured seven at the CF Industries nitrogen production plant in Donaldsonville, LA, a small city on the Mississippi River 10 miles south of Geismar. Several of those injured had burns of sufficient degree that they required transfer to the burn unit in Baton Rouge, LA. Anesthesiologists who work in areas such as these should learn the specific hazards posed by the industries in the area and make specific preparations to manage large numbers of patients who will present with the most likely types of injuries.

Intentional Disasters (Terrorism)

Chemical Agents

Chemicals have been used as weapons for millennia and as weapons of mass destruction for more than 100 years. Their effectiveness has recently been “discovered” by terrorist groups, who have used chemical weapons to attack their perceived enemies. The Aum Shinrikyo terrorist organization poured a nerve agent, sarin, on the floor of subway cars in Tokyo, Japan in 1995, injuring thousands, and had planned the simultaneous release of cyanide gas into the same subway station. Fortunately, the device for creating and releasing the gas malfunctioned. It is not well known, but the cult sent members to Africa in the late 1990s during an outbreak of EVD to bring back virions to be used as a biologic weapon; fortunately, their efforts were unsuccessful.

Nerve Agents

After the sarin gas attack in Tokyo, >5000 persons required emergency medical evaluation, with approximately 1000 manifesting exposure to the nerve agent; 18 people died. Anesthesiologists have a unique understanding of how to manage chemical nerve agents, irreversible anticholinesterases, because they administer a reversible anticholinesterase drug (neostigmine) on a daily basis. The excess acetylcholine that accumulates in cholinergic nerve terminals accounts for the toxicity of the nerve agents. (A cholinergic drug such as glycopyrrolate is administered at the same time as neostigmine to antagonize the muscarinic effects of the excess acetylcholine.) Excess acetylcholine (at preganglionic muscarinic and postganglionic muscarinic and nicotinic receptors) causes copious lacrimal and nasal secretions, miosis, bronchospasm, arrhythmias, and tonic muscle contractions leading to respiratory paralysis. Central nervous system toxicity causes seizures. The combination of status epilepticus and respiratory paralysis results in death.

If several patients arrive simultaneously in an emergency department complaining of shortness of breath and who exhibit rhinorrhea, miosis, and an irregular cardiac rhythm, it is possible that they have been exposed to a nerve agent as a result of a terrorist attack (Table 16.2). The differential diagnosis includes opioid overdose, but opioids do not cause rhinorrhea, bronchospasm, or diarrhea. A mnemonic to help remember unopposed parasympathetic activity is dumbels (D-diarrhea, U-urination, M-miosis, B-bronchorrhea and bronchoconstriction, E-emesis, L-lacrimation, and S-salivation).

Patients who may have been exposed to a nerve agent must undergo decontamination (if that has not already been performed). The primary goals are to remove the nerve agent to prevent further injury and contamination of others. After donning personal protective equipment (PPE), remove the patient's clothes, and if the patient has been exposed to a liquid nerve agent (as opposed to vapor), wash the patient with copious amounts of water in 0.5% hypochlorite (household bleach). The bleach is not as critical as washing with copious amounts of water. The only exception is if the patient is in extremis; treat these patients first and then decontaminate the patient and oneself. Patients who are in respiratory arrest or in status epilepticus should be treated as any other patient with these diagnoses. Antagonize the excess acetylcholine with a cholinergic agent (e.g., atropine at a dose beginning at 0.4 mg and repeated at 5-minute intervals until symptoms and signs have resolved; doses of 1–2 g are sometimes required) intravenously to attenuate and block the muscarinic side effects of the agents. Consider also administering pralidoxime chloride (2-PAM chloride). Pralidoxime chloride is an oxime that reactivates acetylcholinesterase by removing the nerve agent from its binding site on the enzyme. Spontaneous reactivation of acetylcholine esterase is variable and depends on the nerve agent used, the concentration of the agent to which the patient has been exposed, and the amount

Table 16.2 Symptoms and Treatment of Patients with Nerve Agent Exposure

Minimal	Moderate	Severe
Miosis, headache	Severe rhinorrhea	Respiratory failure
Rhinorrhea, salivation	Dyspnea/ bronchospasm	Seizures/flaccid paralysis
Chest tightness	Muscle fasciculations	Incontinence
Remove from exposure	Wet decontamination	Decontaminate/ atropine
Remove clothes	Atropine	2-PAM CL, ventilate

of time that has elapsed since exposure. Therefore, 2-PAM-CL should be administered as soon as possible to a patient if exposure to a nerve agent is suspected.

Pulmonary Agents

Pulmonary agents are gases at room temperature that damage the lungs. Any gas (e.g., otherwise harmless gases such as helium or nitrogen) could be considered a pulmonary agent because if released into a closed space in sufficient volume it could displace O_2 and cause asphyxiation. Chlorine and phosgene, however, are the two classic pulmonary agents, and are most likely to be used by terrorists. Both gases are extremely toxic to the lungs and often cause acute respiratory distress syndrome even if small quantities are inhaled. Treatment is no different than that of silo filler's disease or farmer's lung (caused by exposure to nitrogen dioxide when a farm worker opens or enters a silo that has inadequate ventilation). Management of the resulting noncardiac pulmonary edema from NO_2 or the pulmonary agents is symptomatic: mechanical ventilation using small tidal volumes (6–8 mL/kg) while maintaining peak airway pressures <30 cm H_2O , positive end expiratory pressure and inspired oxygen concentrations of 50%–60% or less.

Blood Agents

The blood agents, hydrogen cyanide and cyanogen chloride, are the third class of chemicals that could be used as WMD. Because of the instability of cyanogen chloride, hydrogen cyanide is more likely to be delivered as an aerosol in a closed environment inducing cyanide toxicity to those who inhale the agent. Cyanide toxicity is an entity with which anesthesiologists are familiar because sodium nitroprusside, if administered at a high-dose for extended periods of time, also causes cyanide toxicity. Cyanide interrupts the electron transport chain in mitochondria, inhibiting aerobic metabolism. Untreated patients die rapidly. Cyanide poisoning is treated with an initial small, inhaled dose of amyl nitrite, followed by intravenous sodium nitrite and then intravenous sodium thiosulfate. The sulfur moiety of the thiosulfate serves as a receptor for the metabolic degradation of cyanide ions to thiocyanate, which is then excreted by the kidneys. Thiocyanate has few side effects until plasma levels exceed 10 mg/dL. Sulfanegen TEA, under investigation as an alternate to thiosulfate, can be administered by intramuscular injection and also converts cyanide into thiocyanate. Hydroxocobalamin, approved in 2008 is effective for the treatment of cyanide poisoning because its cobalt moiety binds cyanide ion rendering it inactive. Depending upon the severity of the poisoning, tracheal intubation, mechanical ventilation with 100% oxygen, and inotropes and vasopressors may be required.

Biologic Agents

Biologic agents are chosen for their ability to cause mass casualties because they are highly contagious or easily distributed over a large geographic area, because of the high morbidity and mortality associated with infection, and because of their ability to cause panic and disruption of normal social behavior. Several agents that fit this description have been used by military forces and terrorists in the past. Category A agents are those that are highly contagious, have a high mortality rate, along with other characteristics that make them ideal WMD (Table 16.3).

Smallpox

The last case of naturally occurring smallpox in the world was reported in 1977 in Somalia and in 1980, the World Health Organization announced that the world was free of this scourge. Routine vaccination for smallpox is no longer carried out, except in the military and for some public health care workers considered at high risk of contracting the disease (i.e., individuals who the government would rely on to staff vaccination stations if there were a breakout). Terrorists might consider using smallpox as weapon because an increasing number of people no longer have immunity. Smallpox is highly infective, with 40% to 80% of non-vaccinated individuals becoming affected after exposure to only 10 to 100 virions. Virions can be transmitted as an aerosol or on clothing or linen from an infected individual. Thirty to fifty percent of infected patients die. Immunity decreases over time in individuals who have been vaccinated, but the vaccine provides some protection even after 20 years.

Table 16.3 Biologic Agents Used as Weapons of Mass Destruction		
Category A	Category B	Category C
<i>Bacillus anthracis</i> (anthrax)	<i>Coxiella brunetti</i> (Q fever)	Encephalitic viruses
<i>Variola major</i> (smallpox)	<i>Vibrio cholerae</i>	
<i>Yersinia pestis</i> (plague)	<i>Burkholderia mallei</i> (glanders)	
<i>Clostridium botulinum</i> (botulism)	Enteric pathogens	
Viral hemorrhagic fever (Ebola)	Cholera, <i>cryptosporidium</i>	
Various biologic toxins		
Smallpox		

Patients who are infected with smallpox present with malaise, headache, and backache with fever to as high as 40° C. The fever decreases over the next 3–4 days at which time the characteristic rash develops. Smallpox has a predilection for the distal extremities and face, although no part of the body is spared, with all lesions at the same stage. If a case of smallpox is identified, the Centers for Disease Control and Prevention has plans to quarantine the patient. That patient's immediate contacts and individuals within the geographic area would be vaccinated. There are stockpiles of vaccines placed strategically throughout the United States just for such an event. The Centers for Disease Control and Prevention and the states' departments of health will implement their quarantine and vaccination plans should an index case or several cases (a cluster) occur.

Anthrax

Anthrax (*bacillus anthracis*) spores clump in the nasopharynx when inhaled. For *B. anthracis* to be used as a weapon, therefore, it must finely ground so that it can be aerosolized, inhaled, and deposited in terminal bronchioles and alveoli. When inhaled, weaponized anthrax has very high infection and fatality rates. For example, one of the letters mailed in the anthrax attacks of 2001 contained 2 g of weapons-grade anthrax. With an LD₅₀ of 1000 spores, under optimum conditions, this was enough material to infect 50 million individuals and cause a fatality rate as high as 80%. The accidental release of anthrax spores at a facility in Sverdlovsk in the former Soviet Union in 1979 killed 66 of the 77 people who were infected (i.e., an 86% mortality rate).

Anthrax presents in a manner similar to an influenza-like disease with fever, myalgias, malaise, and a nonproductive cough that may or may not be associated with chest pain. After a few days, the patient appears to improve, but then develops dyspnea, hemoptysis, stridor, chest pain, and cyanosis. Chest X-ray reveals a widened mediastinum. If untreated, death occurs in 1–2 days. Penicillin G was the treatment of choice before several countries engineered a resistant strain. Immunization against anthrax is undertaken for the US military but requires six subcutaneous injections administered over 2 years. Ciprofloxacin or doxacycline are the recommended antibiotic treatment for a patient with an active case of anthrax.

Plague

Yersinia pestis (bubonic plague) was the cause of the Black Death that killed one-third of the population of Europe in the 14th century. Rodents and fleas are the natural hosts for *Y. pestis*, and the infection is transmitted through fleas. Humans are an accidental host, usually acquiring the disease from a fleabite, although direct person-to-person transmission can occur from patients with

pneumonic plague. The mortality rate for either bubonic or pneumonic plagues is as high as 50%.

After a 2- to 6-day incubation period, an infected patient presents with sudden onset of fever, chills, weakness, and headache. Bubo, intensely painful swelling of the lymph nodes in the groin, axilla, or neck, also develop at this time. Without treatment, patients develop septic shock with cyanosis and gangrene in peripheral tissues. Patients with bubonic plague can seed their lungs with organisms, developing pneumonic plague. Aerosolized secretions caused by coughing can then infect others. The diagnosis can be confirmed with a gram stain or culture of organisms from blood, sputum, or buboes. The treatment of choice is streptomycin, but chloramphenicol or tetracycline can also be used.

Tularemia

Francisella tularensis (tularemia) has some similarity to anthrax and plague, but is not nearly as dangerous. Normally, humans acquire *F. tularensis* with direct contact of an infected animal or from the bite of an infected tick or deerfly. As few as 10 or 50 organisms can invade the body either through hair follicles or abrasions. After an incubation period of 2–6 days, swelling and ulceration is noted at the site of entry. As the swelling continues, the skin eventually breaks, creating an ulcer that develops a necrotic base that becomes black as it scars. *F. tularensis* would most likely be delivered as an aerosol from an airplane. There would then be a 3- to 5-day incubation period after inhalation. The onset of disease would then be marked with fever, pharyngitis, pneumonitis, and hilar lymphadenopathy with a mortality rate of 5% to 15%.

The treatment of choice for tularemia is streptomycin, although gentamicin, tetracycline, and chloramphenicol have been used.

Hemorrhagic Fevers

A number of viral hemorrhagic fevers are listed as Category A agents including the arenaviruses (Lassa fever), bunya viruses (hanta), flaviviruses (Dengue) and filoviruses (Ebola and Marburg). There are at least 18 viruses that cause human hemorrhagic fevers; they are characterized by viral replication in lymphoid cells with incubation between 2 and 18 days. Patients then develop fever ($>38.6^{\circ}\text{C}$), headache, myalgias, abdominal pain, and vomiting, depending on the agent itself and the amount that is inhaled or inoculated across the skin. During the EVD pandemic that began in West Africa in December of 2013 symptoms appeared 8–10 days after an 8-year-old boy handled a bat, the only known reservoir for Ebola virus. Lessons learned in central Africa from the more than 20 epidemics that have occurred there since 1976 were forgotten. Control of the pandemic required implementation of practices that are important for managing any contagious infectious disease, but

especially for any Class A agent. Patients with the disease must be quarantined, contacts must be identified and isolated from the rest of the population, health care workers providing care must follow strict infection control protocols, and safe burial practices must be implemented. Treatment of EVD is supportive and includes maintenance of intravascular volume and oxygenation and treating associated infections. Some patients have recovered more quickly after the intravenous administration of plasma obtained from survivors. Several vaccines are currently being studied.

Radiologic Agents

An industrial accident or the intentional use of radiologic agents by a terrorist group would be the most likely reason that a large population would be exposed to radiation. Terrorists have tried twice to detonate a “dirty bomb,” which is a conventional explosive device surrounded with radioactive material, but fortunately were unsuccessful. Although the use of a dirty bomb is of concern, an accident at a nuclear power plant is far more likely (see Table 6.2). After a nuclear power plant accident, patients are often externally radiated and, may or may not require decontamination and treatment depending on the type and amount of radiation exposure. Assessment may be difficult, but individuals who have no symptoms after 6 hours are unlikely to have received a dose of radiation that requires hospitalization. Those who are symptomatic are hospitalized if possible for serial measurement of white blood cell counts. If the white blood cell count remains stable for 48 hours, the patient may be discharged.

The current policy in the United States is that after any release of radiation, local public health departments will distribute potassium iodide tablets within 24 hours to protect the thyroid of all potentially exposed individuals. Those with the greatest exposure may require hospitalization for treatment of the sequelae of radiation exposure, which include bone marrow failure leading to infection and coagulopathy, gastrointestinal bleeding caused by mucosal damage to the lumen of the intestines, and thrombocytopenia. Treatment of infection, transfusion and volume resuscitation and treatment with G-CSF (granulocyte colony stimulating factor) may be lifesaving.

Nuclear Disasters

A nuclear bomb detonation is very unlikely, but significant planning for the management of such a catastrophe has occurred. Guidelines are in place and are available for use by health care professionals. Most survivors will present with traumatic injuries similar to those seen with conventional explosions. These injuries will be the immediate cause of morbidity and mortality; sequelae of radiation exposure may then appear days to years later depending on the degree of exposure.

Explosive (High Energy) Agents

Detonation of an improvised explosive device (IED) is by far the most widely used weapon by terrorists. On April 15, 2013, at the finish line of the Boston Marathon, two IEDs—pressure cookers loaded with gunpowder, nails, and ball bearings—were detonated, killing three bystanders at the scene (Fig. 16.1). Another 264 runners and bystanders were injured and transported to 29 local hospitals, underscoring what was discussed previously: Casualties do not preferentially go to Level I trauma centers (the greater Boston area has 11 Level 1 adult and pediatric trauma centers). Sixteen patients had traumatic amputations; their limbs were either severed during the explosion or sustained such severe damage that they were not salvageable. Three patients had more than one traumatic amputation. An IED detonation may cause lacerations, thermal injury, multiple penetrating wounds from shrapnel, fractures, blunt soft tissue injury, traumatic amputations, and traumatic brain injury from the primary, secondary, and tertiary blast effects. Although a “dirty” bomb has not yet been detonated by terrorists, this could happen in the future. These patients require decontamination prior to evaluation, stabilization, and treatment, unless the patient has life-threatening injuries that require treatment before decontamination.

Patients with any evidence of burns to the face or airway should be intubated, awake if possible, because a significant number of



Figure 16.1 The finish line at the Boston Marathon as the improvised explosive device exploded.

Photo by Dan Lampariello. Reprinted with permission.

these patients will have mild to moderate glottic edema at the time of intubation. Patients with burns must be managed aggressively with fluid resuscitation. Because these patients will have other injuries from shrapnel and amputations, intravascular volume may be significantly decreased and a very liberal fluid resuscitation policy must be followed. Many patients will require more fluid than is commonly given with either the Parkland or Brook Army Burn Center formulae. In addition to volume resuscitation, forced diuresis with alkalinization of the urine in patients with a crush injury or extensive soft tissue and skeletal muscle damage may be organ and lifesaving. Patients with the most severe injuries are likely to have had significant blood loss and are at risk of developing acute traumatic coagulopathy. Rapid, acute blood loss leads to decreased O_2 delivery because left ventricular end diastolic volume and cardiac output are decreased, and the decreased blood hemoglobin levels results in a decreased arterial O_2 content. Hemorrhagic shock is the end result manifested by hypotension, tachycardia, lactic acidosis, and hypothermia. The latter two predispose the patient to developing acute traumatic coagulopathy. After an incident such as the Boston Marathon bombing, the blood bank should be alerted to the possibility that the hospital's massive transfusion protocol may need to be activated for multiple patients. The operating rooms should be warmed and measures taken to maintain patients' temperature during surgery. Damage control resuscitation is commonly used in these cases, so the infusion of crystalloid should be very limited, with a goal of replacing what was lost using thromboelastography or one of its surrogates to guide blood component therapy. Tranexamic acid has been shown to improve outcome but the improvement in outcome is moderate at best. The best outcomes are achieved by stopping the bleeding as soon as possible by whatever means necessary, for example, by placing a tourniquet in the field or by rapid transport of a patient to the hospital and an operating room for damage control surgery. With planning and preparedness, patients who arrive at a hospital may have a 98% chance of survival.

Conclusion

Few physicians who work in the community or at an academic center are experienced in the management of mass casualties. However, one can prepare for one by developing a family care plan, a personal care plan, and a professional plan that includes a review of the anesthesiology department's emergency response plan and

knowing where to find information that would be of great importance in managing patients injured by WMD.

Further Reading

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Chapter 17

Ethical Considerations

Robert B. Schoenberger and Stanley H. Rosenbaum

Informed Consent 442

Organ Harvesting and the Declaration of Brain
Death 443

Refusal of Blood Transfusion 445

Informed Consent

Definition

The key components of informed consent are 1) an understanding of what the patient is consenting to, 2) an understanding of the major risks and benefits, and 3) an understanding of the alternatives to the proposed care.

Presentation

- As a general rule, informed consent is required prior to all invasive procedures.
- In an emergency in which no decision maker is available, physicians should act in accordance with what a reasonable person would consider to be in the best interests of the patient.

As both a legal and ethical matter, informed consent must be obtained prior to the performance of any invasive procedure.

- Competent patients may refuse to give consent for any procedure for any reason.
- For competent patients, informed consent must be obtained from the patient and must explain the purpose of the proposed procedure as well as the major risks, benefits, and alternatives to the procedure.
- In the case of incompetent patients who were formerly competent, a surrogate decision maker must be sought. The essential elements and requirements of the informed consent process are the same when dealing with surrogates as with patients themselves. The surrogate's role is to act in substituted judgment, that is, to authorize what the patient's wishes would be if the patient were in a position to express them.
- In the case of a minor child or individual who has never been competent, informed consent must be obtained from the legal guardian or conservator whose role is to represent the best interests of the incompetent person.
- In an emergency for which delay would result in the loss of life or limb or cause serious morbidity, and in which a competent decision maker cannot be located, physicians have an ethical duty to act in accordance with what a reasonable person would believe is in the best interest of the patient until informed consent can be sought.

Exceptions to the Need for Informed Consent

- Patients may be incapable of informed consent due to acute injury, chronic brain disease, medication, intoxication, mental impairment, mental illness, or young age. In all of these cases, the physician's obligation to obtain informed consent does

not go away; the process is transferred from the patient to the surrogate decision maker.

- Physical disability or language barrier may make obtaining consent inconvenient, but has no impact on a patient's competency or on the obligation of the caregiver to obtain consent from the patient.
- An emergency exemption to obtaining informed consent can be considered to be a reasonable presumption of consent. If consent is later withdrawn by the patient or a surrogate, the emergency exemption no longer applies.
- In the case of minor children, some treatments may be mandated by the courts despite the parents' refusal to give informed consent. Keeping in mind the emergency exemption, it is prudent for a care provider to seek institutional administrative support in such situations.

Further Reading

For a discussion of the typology of informed consent as distinct from shared decision making and simple consent, see:

Whitney SN, McGuire AL, McCullough LB. A typology of shared decision making, informed consent, and simple consent. *Ann Intern Med.* 2004; 140(1): 54–59.

Organ Harvesting and the Declaration of Brain Death

The Ethical Standard of Care

- Outside the domain of the consenting living donor, organ harvesting may occur after either cardiac or brain death.
- The declaration of brain death in the United States requires documentation that the entire brain, including the brain stem, has permanently ceased to function.
- The declaration of brain death must generally be made by at least one physician who is not connected to the transplantation process.
- Comatose patients with residual brain function, including persistently vegetative patients, do not meet the legal definition of brain death and may not undergo organ harvesting unless a donation after cardiac death has been arranged.
- A patient who shows evidence of spontaneous respiration or other brain stem activity is not legally dead in the United States.

Determination of Brain Death

- Reversible causes of apparent coma must be ruled out both by history and physical examination. Severe metabolic derangement, intoxication, hypothermia, and residual neuromuscular blockade must be considered and, where appropriate, treated.
- The clinical determination of brain death requires:
 - Unresponsiveness, including to painful stimuli in cranial nerve territories
 - The absence of all brain stem reflexes
 - Apnea in response to a hypercarbic challenge
- Most US jurisdictions hold that the clinical determination of brain death is sufficient in adults for the determination of death without the need for further neurophysiologic testing.
- If in doubt, or if clinical tests for the determination of brain death cannot be performed, diagnosis of brain death may also be based on an electroencephalogram showing absent brain activity, or perfusion studies that demonstrate the absence of brain blood flow.
- Legal and institutional guidelines vary but may require that the determination of brain death be performed by more than one physician or at more than one point in time.
- Some United States jurisdictions allow for the next of kin to refuse that the medical determination of brain death be used as the basis to declare the death of the patient.

According to the American Academy of Neurology, brain death may be diagnosed even if any of the following are present:

- Spontaneous movements of limbs other than pathologic flexion or extension
- Intercostal expansion without significant tidal volumes
- Sweating, blushing, or tachycardia
- Hemodynamic stability
- Absence of diabetes insipidus (i.e., preservation of hypothalamic function)
- Deep tendon reflexes
- Babinski reflex

Donation after Cardiac Death

Donation after cardiac death (DCD) refers to the practice of removing a severely ill patient from active life support in the expectation that the patient will have a cardiac arrest within a few minutes. This is distinct from donation after brain death, in which asystole is not required. A DCD donor is pronounced dead after asystole has lasted for a specified period (usually 5 minutes). The transplant

team then removes organs, generally just the liver and kidneys, for transplantation. This procedure must satisfy strict constraints:

- The patient's surrogate decision makers must have given formal consent for withdrawal of active life support.
- To avoid any potential conflicts of interest, there must be a sharp separation between the medical team caring for the patient and the transplant team.
- A formal institutional protocol must describe the details of the process, including the acceptable participants, the venue for the withdrawal of active support, the allowed time for the patient to expire after withdrawal of life support, and the period of asystole needed for the declaration of death.
- If the patient does not become apneic and pulseless during the waiting time after withdrawal, generally 1 hour, the patient is returned to regular hospital care as may be appropriate for the patient.

Further Reading

American Academy of Neurology, Quality Standards Subcommittee. Practice parameters: determining brain death in adults, *Neurology*. 1995; 45: 1012–1014.

Van Norman GA. A matter of life and death: what every anesthesiologist should know about the medical, legal, and ethical aspects of declaring brain death. *Anesthesiology*. 1999; 91(1): 275–287.

445

Refusal of Blood Transfusion

The Ethical Standard of Care

- Competent adult patients may refuse blood transfusion or any other treatment for any reason, even if withholding such treatment may cause death.
- Patients should be asked before their surgery whether they have any objections to receiving blood products. If the patient denies the use of blood products, his or her wishes should be honored.
- Individual patient preferences may differ regarding cell saver systems and extracorporeal blood circuits. The specifics of a patient's preferences should be clarified prior to surgery.
- A care provider who objects to withholding appropriate blood products may defer care to a colleague who is able to honor the patient's autonomy. In cases in which alternative providers do not exist, physicians may have an obligation to provide care despite their own objections.

Exceptions to the Refusal of Blood Transfusion

- United States case law generally holds that parents of preadolescent children may *not* refuse blood transfusions on behalf of their minor children.
- Older adolescent children with objections to blood transfusion fall into a gray area in which an ethics consult should be requested.
- Gravid patients with viable fetuses present another ethical dilemma for which conflicting case law exists, and for which an ethics consult may be warranted.

Treatment Modalities for Patients Who Refuse Transfusion

- A variety of techniques may reduce risk to patients who refuse blood transfusion.
- Before surgery, patients may be given recombinant erythropoietin in an effort to increase hemoglobin concentration. Some insurance carriers will not pay for this treatment without sustained advocacy on behalf of the patient.
- Occasionally, patients who refuse anonymous-donor banked blood may still consent to autologous blood banking.
- Patients who refuse autologous blood banking may sometimes consent to the withdrawal of blood if it is kept in a continuous circuit with the body. Such patients may undergo intraoperative withdrawal of blood into a blood donation bag that is left connected to the IV circuits. The blood can be slowly reinfused until a transfusion is needed.
- Deliberate hemodilution techniques are commonly used, but evidence-based guidelines have yet to be developed.
- Steps to reduce blood loss include modifications of surgical technique, controlled hypotension, use of ddAVP, use of anti-thrombolytics, and use of recombinant Factor VIIa.
- Cell saver systems can reduce the need for transfusion if patients consent to them. In situations, such as oncologic surgery, where cell saver is normally contraindicated, surgical blood loss may still be collected in a cell saver system. This blood should then be discarded unless the need for blood becomes critical.
- Patients also may be given recombinant erythropoietin after surgery. There are also case reports of salvage therapy using

hypothermia, sedation, and neuromuscular blockade to reduce oxygen consumption in patients with otherwise fatal anemia.

Further Reading

For a discussion of some relevant case law, see:

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Index

A

- Abdominal aortic aneurysm, ruptured, 318–321
- Abdominal compartment syndrome, 312
- Abscess, epidural, from regional anesthesia, 360–362
- Acetaminophen
 - for postoperative pain, severe, 352
 - for thyroid storm, 168
 - for trauma, pediatric, 279
- Acetazolamide, for alkalosis, 132
- Acidosis, 126–128
- Acute intermittent porphyria, 165
- Acute lung injury (ALI), 36–37
- Acute renal failure, postoperative, 339–341
- Acute respiratory distress syndrome (ARDS), 36–37
- Acute transfusion reaction, 282–283
- Adenosine, for tachycardia
 - narrow complex, 66, 67
 - supraventricular, unstable pediatric, 272
- Adrenal insufficiency, acute, 128–130
- Advanced life support, pediatric non-OR, 269–274
- Advanced Trauma Life Support (ATLS), pediatric, 276–277
- Air embolism
 - in pregnancy, 198–202
 - venous, 187–189
- Airway emergencies, 7–34
 - airway fire, 8–9
 - aspiration, 9–10
 - cannot intubate/ cannot ventilate, 16–17
 - cannot intubate/ can ventilate, 15–16
 - endotracheal tube ventilation difficulty, 18–21
 - hemoptysis, 21–24
 - laryngospasm, 29–30
 - Ludwig's angina, 30–31
 - mask ventilation difficulty, 18
 - rapid-sequence intubation, 32–34
 - surgery- and anesthesia-related, 10–13
 - tonsillectomy, bleeding after, 13–14
 - tracheal extubation high-risk, 24–27
 - upper airway obstruction, intrinsic, 27–29, 28f
- Airway exchange catheter (AEC)
 - for tracheal extubation, high-risk, 26–27
 - for tracheal injury, 124
- Airway fire, 8–9
- Airway obstruction
 - bronchial, from intrathoracic lesions, 108–112
 - pediatric
 - complete, 260–262
 - lower airway, 247–250
 - upper, intrinsic, 27–29, 28f
- Airway pressure
 - increased, in one-lung ventilation, 117–118
- Airway pressure release ventilation (APRV)
 - for acute lung injury, 36
 - for acute respiratory distress syndrome, 36
 - for difficult controlled ventilation, 43
- Albuterol
 - for airway obstruction, complete pediatric, 261
 - for anaphylaxis, 134
 - for asthma, pediatric, 248, 249
 - for bronchospasm, 38
 - in anaphylaxis, pediatric, 245
 - with near-drowning, 264
 - pediatric, 248
 - for endotracheal tube ventilation difficulty, 21
 - for surgery- or anesthesia-related emergencies, 12
 - for ventilation, difficult controlled, 42
- Alkalosis, 131–132
- Altered mental status, postoperative, 330–332
- Amiodarone
 - for atrial fibrillation, 62
 - for pediatric advanced life support, 271
 - for tachycardia
 - narrow complex, 67
 - supraventricular, unstable pediatric, 272
 - wide complex, 70, 71
 - for ventricular fibrillation, 69
- Amniotic fluid embolism, 198–202
- Amyl nitrite
 - for cyanide poisoning, 433

- Amyl nitrite (Cont.)
 methemoglobinemia from, 256
 Anaphylaxis, 133–135
 from amniotic fluid embolism, 198
 bronchospasm from, 19, 20, 21, 38
 from colloid solutions, 212
 hypotension in, refractory, 345
 pediatric, 244–247
 upper airway obstruction from, 28
 Anesthesia-related emergencies, 10–13
 Anesthetic toxicity, local, 367–370
 in pregnancy, 213–215
 Anhematin, for porphyria, 167
 Anion gap, 127
 Anterior spinal artery syndrome, 178
 Anthrax, 435
 Antidiuretic hormone (ADH), 136
 Aortic dissection, thoracic, 87–89
 Aortic regurgitation, 89–90
 Aortic stenosis, 91–92
 Apgar score, 265t
 Arrhythmias, 60–71
 asystole, 60–61
 atrial fibrillation, 61–63
 bradycardia, 64–65
 tachycardia
 narrow complex, 66–67
 wide complex, 69–71
 ventricular fibrillation, 68–69
 Aspiration, 13
 Aspiration emergencies, 9–10
 Aspirin
 for chest pain, 334
 for myocardial ischemia, 82
 postoperative, 338
 for pregnancy, with pre-eclampsia history, 229
 Assessment, constant, crisis resource, 4
 Asthma, pediatric, 247–250
 Asystole, 60–61
 Atrial fibrillation, 61–63
 Atropine
 for asthma and bronchospasm, pediatric, 248
 for asystole, 60
 for bradycardia, 64, 65, 246
 for cholinergic nerve agents, 432, 432t
 for pediatric advanced life support, 271, 274
 Autonomic hyperreflexia or dysreflexia, 174–175
 Auto-PEEP, 43
 Awake intubation, 396–398
 Awakening, delayed, 330
 Axonal loss, 373
- B**
Bacillus anthracis, 435
 Beck's triad, 72
 Beractant, for neonatal resuscitation, 267
 Bicarbonate. See Sodium bicarbonate
 Biologic agents (bioterrorism), 434–437, 434t
 anthrax, 435
 hemorrhagic fevers, 436–437
 plague, 435–436
 respiratory protection for, 57–58, 57f
 smallpox, 434–435
 tularemia, 436
 Biological disasters, 428–429
 Bisphosphonate, for hypercalcemia, 141
 Black Death, 435–436
 Bleeding (hemorrhage)
 after carotid endarterectomy, 302–304
 after thyroid surgery, 304–306
 massive, 313–315
 in mediastinoscopy, major, 112–114
 in obstetrics, maternal, 219–222
 postoperative, 84–85
 Blood agents, 433
 Blood transfusion refusal, 445–447
 Bone cement implantation syndrome, 284–285
 Bradycardia, 64–65
 fetal, 205–210, 206f, 208f
 Brain death, declaration of, 443–445
 Brain injuries, traumatic, 184–186
 Breathing circuit malfunction, 376–378
 Breech presentation, 195–197
 Bronchial blocker (BB), 407
 for hemoptysis, 23
 in one-lung ventilation, 407–409
 Bronchial obstruction, from intrathoracic lesions, 108–112
 Bronchopleural fistula, 100–102
 Bronchospasm
 adult, 20, 38–39
 with near-drowning, 264
 pediatric, 245, 247–250
 Brown-Sequard syndrome, 178
 Bubonic plague, 435–436
 Bupivacaine
 for complete spinal anesthesia, unintentional, 359
 local anesthetic systemic toxicity from, 367–370
 Burns
 adult, 285–287
 glove and stocking, 254
 pediatric, 250–256
 types of, 250, 285–286
 Butyropheneone, for altered mental status, postoperative, 331
- C**
 Calcitonin, for hypercalcemia, 141

- Calcium chloride
 - for hyperkalemia, 145
 - for hypermagnesemia, 149
 - for hypocalcemia, 143–144
- Calcium, ionized, for hypocalcemia, 143–144
- Candesartan, for congestive heart failure, 76
- Cannot intubate/cannot ventilate, 16–17
- Cannot intubate/can ventilate, 15–16
- Cannula cricothyroidotomy, 399–400
- Carbon monoxide poisoning, 274
 - pediatric, 251, 255–256
- Cardiac arrest
 - in children, 273–274
 - in obstetrics, maternal, 216–218
- Cardiac herniation after pneumonectomy, 103–105
- Cardiac obstruction, from intrathoracic lesions, 108–112
- Cardiac tamponade, 72–73
 - from chest tube occlusion, 85
 - hemodynamic instability with minor blood loss from, 114
- Cardiac trauma, 74–75
- Cardiogenic shock, 310. *See also Shock*
- Cardiovascular
 - emergencies, 59–98
 - arrhythmias, 60–71
 - asystole, 60–61
 - atrial fibrillation, 61–63
 - bradycardia, 64–65
 - tachycardia, narrow complex, 66–67
 - tachycardia, wide complex, 69–71
 - ventricular fibrillation, 68–69
 - cardiac tamponade, 72–73
 - cardiac trauma, 74–75
 - congestive heart failure, 75–77
 - hemorrhage, postoperative, 84–85
 - hypertension, 77–79
 - hypotension, 79–81
 - myocardial ischemia, 81–83
 - pulmonary embolism, 54–56, 85–87
 - thoracic aortic dissection, 87–89
 - valvular disease, 89–96
 - aortic regurgitation, 89–90
 - aortic stenosis, 91–92
 - mitral regurgitation, 93–94
 - mitral stenosis, 94–96
 - venous gas embolism, 96–98
- Cardioversion
 - for atrial fibrillation, 62–63
 - for mitral stenosis, 95
 - for tachycardia narrow complex, 66
 - supraventricular, unstable pediatric, 272
 - wide complex, 70, 71
 - for ventricular fibrillation, 67
- Carotid endarterectomy, bleeding after, 302–304
- Catheter
 - airway exchange for high-risk tracheal extubation, 26–27
 - for tracheal injury, 124
 - femoral vein, 403–405
- Cauda equina syndrome, 179
- Central cord syndrome, 179
- Central venous access, ultrasound-guided, 418–420
- Chemical agents, 431
- Chest pain, postoperative, 332–334
- Chloramphenicol
 - for plague, 436
 - for tularemia, 436
- Chlorine, 433
- 2-Chloroprocaine, for urgent delivery, 209
- Cholinergic drug, 431
- Chvostek sign, 142
- Cinacalcet, for hypercalcemia, 141
- Ciprofloxacin, for anthrax, 435
- Clopidogrel, for myocardial ischemia, 82
- Coma, myxedema, 160–162
- Communication, effective, 4
- Compartment syndrome, abdominal, 312
- Complete breech, 195
- Complete spinal anesthesia, from regional anesthesia, 358–360
- Congestive heart failure, 75–77
- Consent, informed, 442–443
- COX-2 inhibitors, for severe postoperative pain, 352, 353
- Crashes, 429
- Cricothyroidotomy, 399–401
 - cannula (needle), 399–400
 - large-bore (Melker), 400
 - special considerations, 401
- Crisis resource management, 2–6
 - assessment in, constant, 4
 - checklists for, 4, 5b
 - communication in, effective, 4
 - key principles of, 2–3, 2b–3b
 - leader role in, 3
 - scope and aim of, 2
 - team member roles in, 3–4
 - training in, 5
 - triple threat model for, 5–6, 5b–6b
- Critically ill patient laparotomy in, 309–312
 - transport of, 413–415
- Croup, 257t
- Cushing's triad, 175

Cyanide gas poisoning, 431
Cyanogen chloride, 433

D

Damage-control resuscitation, 280

Damage control surgery, in critically ill, 312

Dantrolene, sodium, for malignant hyperthermia, 159, 160

Declaration of brain death, 443–445

Delayed awakening, 330

Delayed hemolytic transfusion reactions, 283

Dengue fever, 436–437

Dental trauma, 287–290

Desflurane, on asthma and bronchospasm, 248

Desmopressin acetate (DDAVP)

for normovolemic hyponatremia with diabetes insipidus, 152

for polyuria in diabetes insipidus, 136

Dexamethasone

for adrenal insufficiency, acute, 129–130

for airway obstruction, complete pediatric, 261

for anaphylaxis, pediatric, 246

for asthma and bronchospasm, pediatric, 248

for endotracheal tube ventilation difficulty, 21

for rigid bronchoscopy, prolonged, 107

for stridor, pediatric postextubation, 275

for thyroid storm, 168

for upper airway obstruction, intrinsic, 29

Dexmedetomidine

for awake intubation, 397

for pain with burn injuries, pediatric, 254

for postoperative delirium prevention, 331–332

Diabetes insipidus, 135–137

Diabetic ketoacidosis, 137–140

Digoxin, for atrial fibrillation, 62

Diltiazem for atrial fibrillation, 62 for tachycardia, narrow complex, 66, 67

Diphenhydramine, for anaphylaxis adult, 134

pediatric, 246

Dirty bomb, 437

Disaster management, 421–440

overview of, 422–423 pre-disaster planning in, 423–425

family disaster plan in, 423–424, 424t

personal emergency preparedness plan in, 424

professional emergency preparedness in, 424–425

response to disaster in, 425–439 (See also Disaster response)

scope of, 422

Disaster preparedness, 422

Disaster prevention, 422

Disaster recovery, 422

Disaster relief, 422

Disaster response, 425–439

for explosive (high energy) agents, 438–439, 438f

for intentional disasters (terrorism), 431–437

biologic agents, 434–437, 434t (See also Biologic agents)

blood agents, 433

chemical agents, 431

nerve agents, 431–433, 432t pulmonary agents, 433

radiologic agents, 437

mass casualty events in, 422–423, 425, 426t

for natural disasters, 426–429

biological, 428–429 geological, 427–428

meteorological, 426–427

for nuclear disasters, 437

for unintentional disasters, 429–431

crashes, 429

fires, 429–430

industrial, 430–431

Distributive shock, 310.

See also Shock

Dobutamine, for

postoperative oliguria/acute

renal failure, 341

Donation after cardiac death (DCD), 444–445

Dopamine

for bradycardia, 65

for oliguria/acute renal failure,

postoperative, 341

for pediatric advance life support, 273

Double crush

phenomenon, 372

Double-lumen

endotracheal tube (DLET),

401–403

Double lumen tube

(DLT), for

hemoptysis, 23

Doxycycline, for anthrax, 435

Droperidol, for

postoperative nausea and vomiting, 347

Drug extravasation, 290–292

Dural puncture,

accidental, in pregnancy,

192–194

Dysreflexia, 174–175

E

- Earthquakes, 427–428
- Ebola viral disease (EVD)
 - as natural disaster, 428–429
 - as terrorism, 436–437
- Ectopic pregnancy,
 - ruptured, 322–324
- Edema, pulmonary, 52–54
- Electric power failure, 379–382
- Embolism
 - air
 - in pregnancy, 198–202
 - venous, 187–189
 - in obstetrics, 198–202, 216
 - pulmonary, 54–56, 85–87
 - venous gas, 96–98
- Enalapril, for congestive heart failure, 76
- Endobronchial intubation,
 - inadvertent, 19–20
- Endotracheal tube. *See also specific topics*
 - difficult ventilation with, 18–21
 - double-lumen, 401–403
 - for neonates, 268, 268t
- Endovascular repair (EVAR), of
 - abdominal aortic aneurysms, 320–321
- Ephedrine
 - for bone cement implantation syndrome, 284
 - for chest pain, postoperative, 334
 - for complete spinal anesthesia, unintentional, 359
 - for hemoptysis, 23
 - for hypotension, 80
 - fetal bradycardia, 207, 210
 - with hemorrhage in mediastinoscopy, 113
 - from neurogenic shock, 180
 - postoperative, 344
 - in pregnancy, 211, 212
 - in pregnancy,
 - total/high spinal anesthesia in, 236
 - for intracranial hypertension, 176
- Epidural abscess, from
 - regional anesthesia, 360–362
- Epidural hematoma,
 - from regional anesthesia, 363–365
- Epiglottitis
 - for asthma and bronchospasm, pediatric, 248
 - pediatric, 256–259, 257t
 - thumb sign, 28, 28f
- Epinephrine
 - for airway obstruction, complete
 - pediatric, 261
 - for anaphylaxis
 - adult, 134
 - pediatric, 245, 246
 - for asystole, 60
 - for bradycardia, 65
 - for bronchospasm, 38
 - for complete spinal anesthesia, unintentional, 359
 - for endotracheal tube ventilation difficulty, 21
 - for hemorrhage in mediastinoscopy, 113
 - for hypotension, 80
 - from cardiac herniation, 104
 - postoperative, 344
 - in total/high spinal anesthesia in pregnancy, 236
 - for laparotomy in critically ill, 310
 - with local anesthetics, 370
 - for pediatric advanced life support, 271, 274
 - for resuscitation, neonatal, 266
 - for rigid bronchoscopy, prolonged, 107
 - for sepsis, puerperal, 230–232
 - therapeutic index of, 246
 - for upper airway obstruction, intrinsic, 29
 - for ventricular fibrillation, 67
- Equipment failure,
 - before anesthesia induction, 382–383
- Equipment problems, 375–391
 - before anesthesia induction, 382–383
 - breathing circuit malfunction, 376–378
 - electric power failure, 379–382
 - network failure, 383–384
 - oxygen pipeline failure, 385–387
 - ventilator failure
 - after anesthesia induction, 388–391
- Erythropoietic porphyria, 165
- Erythropoietin,
 - recombinant, for blood transfusion refusal, 446–447
- Esmolol
 - for congestive heart failure, 76
 - for hypertension, postoperative, 342
 - for myocardial ischemia, postoperative, 338
 - for pheochromocytoma, 163
 - for tachycardia, narrow complex, 66
 - for thoracic aortic dissection, 87
 - for thyroid storm, 168
- ETCO₂ decreased,
 - intraoperative, 40–41

- Ethical considerations, 441–447
 - informed consent in, 442–443
 - organ harvesting and declaration of brain death in, 443–445
 - refusal of blood transfusion in, 445–447
- Etomidate
 - for complete spinal anesthesia, unintentional, 359
 - for laparotomy in critically ill, 310
 - for rapid-sequence intubation, 32, 33
- Explosive (high energy) agents, 438–439, 438f
- Extinguishers, fire, 300
- Extracorporeal membrane oxygenation (ECMO)
 - for acute lung injury/acute respiratory distress syndrome, 37
 - for asthma and bronchospasm, 248
 - for biological disasters, 429
 - for difficult controlled ventilation, 43
 - for hemoptysis, 24
 - for hypoxemia
 - intraoperative, 49
 - one-lung ventilation, 116
- Extravasation, drug, 290–292
- Extubation, tracheal, high-risk, 24–27
- Eye protection, 57
- F**
 - Facial trauma, 307–309
 - Factor VII, recombinant activated
 - for hemoptysis, 24
 - for massive hemorrhage, 315
 - Family disaster plan, 423–424, 424t
 - Famotidine, for pediatric anaphylaxis, 246
 - Femoral vein catheter, 403–405
 - Fenoldopam
 - for pheochromocytoma, 163
 - for porphyria, 166
 - Fentanyl
 - for awake intubation, 397
 - for burns, 252
 - for pain with burn injuries, pediatric, 254
 - for rapid-sequence intubation, 33
 - Fetal bradycardia, 205–210, 206f, 208f
 - Fetal heart rate, 210
 - Fetal heart rate patterns, 205–207, 206f, 208t
 - Fibrillation
 - atrial, 61–63
 - ventricular, 68–69
 - Fire. *See also* Burns
 - airway, 8–9
 - injuries from, 429–430
 - operating room, 298–300
 - Fire extinguishers, 300
 - Fisher grading scale, subarachnoid hemorrhage, 182
 - Fluid shields, 57
 - Flumazenil
 - for altered mental status, 331
 - for hypoxia, postoperative, 336
 - for respiratory depression/failure, postoperative, 351
 - Footling breech, 195
 - Foreign body, inhaled
 - adult, 105–107
 - pediatric, 257t, 260
 - 4 Ds, 256
 - Francisella tularensis*, 436
 - Frank breech, 195
 - Free water deficit, 153
 - Furosemide
 - for acute transfusion reaction, 282
 - for congestive heart failure, 76
 - for hypercalcemia, 141
 - for hypervolemic hypernatremia, 152
 - for hyponatremia, 154
 - for hypoxia, postoperative, 336
 - for oliguria/acute renal failure, postoperative, 340
 - for pulmonary edema, 53
 - in difficult controlled ventilation, 42
 - for transurethral resection of the prostate syndrome, 170
- G**
 - Gabapentin, for neuropathic pain from peripheral nerve injury, 372
 - Gas embolism, venous, 96–98
 - Gastrointestinal bleeding, upper, 324–327
 - Gentamicin, for tularemia, 436
 - Geological disasters, 427–428
 - Globe injury, from regional anesthesia, 366–367
 - Glove and stocking burns, 254
 - Glucagon
 - for anaphylaxis with beta-blockers, 134
 - for bradycardia from beta-blockers, 65
 - in diabetic ketoacidosis, 138
 - in hypermetabolism, 251
 - Glycopyrrolate
 - for asthma and bronchospasm, pediatric, 248
 - for hypoxia, postoperative, 336
 - for nerve agents, 431
 - Goggles, 57
 - Gown, 57

H

- H1N1 influenza,
respiratory
protection for,
57, 57f
- Haldol, for postoperative
altered mental
status, 331
- Hanta virus, 436–437
- Headache, postdural
puncture, 194
- Health care worker
occupational
exposure,
296–298
- Heart failure, congestive,
75–77
- Heart rate, fetal, 210
- Heart rate patterns,
fetal, 205–207,
206f, 208t
- Hematoma, epidural,
from regional
anesthesia,
363–365
- Hemin, for porphyria, 166
- Hemolytic transfusion
reactions,
delayed, 283
- Hemoptysis, 21–24,
44–46
- Hemorrhage. *See*
Bleeding
(hemorrhage)
- Hemorrhagic fevers,
436–437
- Hemorrhagic shock,
313–315. *See also*
Shock
laparotomy in critically
ill for, 310
from upper
gastrointestinal
bleeding, 324
- Heparin
for chest pain,
postoperative, 333
for deep venous
thrombosis
prophylaxis,
postsurgical, 334
for drug extravasation,
293
for hypoxia from
pulmonary edema,
postoperative,
336
for myocardial
ischemia, 82
- in post-carotid
endarterectomy
bleeding, 302, 303
protamine reversal of,
85, 304
for pulmonary
(thrombo)
embolism, 55, 86
prevention of, 56
for thromboembolism
in pregnancy, 199,
200
prevention of, 201
- Hepatitis B immune
globulin, 297
- Hepatitis B vaccine, 297
- Heterotopic pregnancy,
324
- Hydralazine
for hypertension, 78
postoperative, 342
for mitral regurgitation,
93
reflex tachycardia
from, 164
- Hydrochloric acid,
for metabolic
alkalosis, 132
- Hydrocortisone
for adrenal
insufficiency,
acute, 129–130
for anaphylaxis, 134
for bronchospasm, 39
for hypotension
from adrenal
insufficiency, 81
for thyroid storm, 168
- Hydrogen cyanide
poisoning, 433
adult, 286, 287
pediatric, 251, 252,
255–256
- Hydromorphone, for pain
in burn injuries,
pediatric, 254
severe postoperative,
352, 353
- Hydroxocobalamin, for
hydrogen cyanide
poisoning
adult, 433
pediatric, 252
- Hypercalcemia, 140–142
- Hypercarbia,
intraoperative,
46–48
- Hyperglycemia,
hyperosmolar,
137–140
- Hyperkalemia, 144–146
- Hypermagnesemia,
148–149
- Hypermetabolism
in burn injuries,
pediatric, 250, 251
in hypercarbia, 46
in malignant
hyperthermia,
158–160
in thyroid storm,
167–169
- Hypernatremia, 151–153
- Hyperosmolar
hyperglycemia,
137–140
- Hypertension, 77–79
intracranial, 175–178
postoperative,
341–343
in pre-eclampsia,
226–230
in pregnancy, 227
pulmonary, with
amniotic fluid
embolism, 201
- Hypertensive
hypovolemic
hemodilution,
for subarachnoid
hemorrhage, 183
- Hypocalcemia, 142–144
- Hypokalemia, 146–148
- Hypomagnesemia,
150–151
- Hyponatremia, 153–155
- Hypotension, 79–81, 210
ephedrine for, 113,
180, 236
epinephrine for, 104,
236
from ketamine in
critically ill, 310
maternal, in
pregnancy,
210–213
phenylephrine for, 91,
104, 113, 169,
179–180
postoperative,
343–345
- Hypothermia, 155–158.
See also Shock
with burn injuries,
pediatric, 251
in hemorrhage,
massive, 280
in laparotomy in
critically ill, 310,
311, 312

- Hypothermia (cont.)
 in near-drowning, 262, 264
 prevention of,
 in neonatal
 resuscitation, 267
 in trauma, 277
 Hypovolemic shock, 310.
See also Shock
 Hypoxemia. *See also*
specific disorders
 intraoperative, 48–50
 in one-lung ventilation,
 115–117
 Hypoxia, postoperative,
 335–337
- I**
- Improvised explosive
 device (IED),
 438–439, 438f
 Industrial disasters,
 430–431
 Influenza, 428–429
 Informed consent,
 442–443
 Inhaled foreign body
 adult, 105–107
 pediatric, 257t, 260
 Insulin
 for diabetic ketoacidosis
 and hyperosmolar
 hyperglycemia,
 139
 for hyperkalemia, 145
 Intensive care unit,
 transfer of patient
 care in, 415
 Intentional disasters
 (terrorism),
 431–437
 biologic agents,
 434–437, 434t
 (See *also* Biologic
 agents)
 anthrax, 435
 hemorrhagic fevers,
 436–437
 plague, 435–436
 smallpox, 434–435
 tularemia, 436
 blood agents, 433
 chemical agents, 431
 nerve agents, 431–433,
 432t
 pulmonary agents, 433
 radiologic agents, 437
 Intra-arterial injection,
 292–294
- Intracranial hypertension,
 175–178
 Intrathoracic lesions,
 obstruction from,
 108–112
 Intubate
 cannot/cannot
 ventilate, 16–17
 cannot/can ventilate,
 15–16
 Intubating laryngeal masks
 (ILMs), 405–407
 Intubation
 awake, 396–398
 endobronchial,
 inadvertent, 19–20
 failed
 after initial attempt,
 15–16
 in pregnancy,
 202–205
 rapid-sequence, 32–34
 retrograde, 409–411
 lpratropium, for
 endotracheal
 tube ventilation
 difficulty, 21
 Isoflurane, for
 endotracheal
 tube ventilation
 difficulty, 20, 21
- K**
- Ketamine
 for aortic regurgitation,
 89
 for asthma, pediatric,
 248
 for bronchospasm
 in anaphylaxis,
 pediatric, 245
 pediatric, 248
 prevention of, 39
 for cardiac tamponade,
 73
 for complete spinal
 anesthesia,
 unintentional, 359
 for emergency
 treatment, 428
 for endotracheal
 tube ventilation
 difficulty, 21
 hypotension from
 in critically ill, 310
 postoperative, 345
 for hypovolemia and
 hemodynamic
 instability, 85
 for pain with burn
 injuries, pediatric,
 254
 for rapid-sequence
 intubation, 33
 for trauma, pediatric,
 279
 Ketoacidosis, diabetic,
 137–140
 Kussmaul breathing, 126
- L**
- Labetalol
 for chest pain,
 postoperative,
 334
 for hypertension, 78
 intracranial, 176
 postoperative, 342,
 343
 for myocardial
 ischemia, 82
 postoperative, 338
 for pheochromo-
 cytoma, 163, 164
 for subarachnoid
 hemorrhage, 182
 for thoracic aortic
 dissection, 87
 for thyroid storm, 168
 Laparotomy, in critically ill
 patient, 309–312
 Laplace, Law of, 319
 Large-bore cricothyroid-
 otomy, 400
 Laryngeal masks,
 intubating,
 405–407
 Laryngospasm, 29–30
 Laryngotracheo-
 bronchitis, 257t
 Lassa fever, 436–437
 Latex allergy, pediatric,
 246
 Law of Laplace, 319
 Levetiracetam, for seizure
 prevention in
 intracranial
 hypertension, 177
 Levothyroxine, for
 myxedema coma,
 161
 Lidocaine
 with airway exchange
 catheter, 26, 27
 for awake intubation,
 397–398
 for bronchospasm
 prevention, 39

- for carotid endarterectomy, 65
 - for delivery, urgent, 209
 - for drug extravasation, 293
 - for femoral vein catheterization, 404
 - for pediatric advanced life support, 274
 - for rapid-sequence intubation, 33
 - for retrograde intubation, 410
 - for right internal jugular central line placement, 419
 - for tachycardia, wide complex, 70
 - toxicity of, 367–370
 - for transvenous pacing, 416
 - for ventricular fibrillation, 69
 - Lidocaine with epinephrine
 - test dose
 - for accidental dural puncture diagnosis, 192
 - in pregnancy, 215, 218, 237
 - spinal, 360
 - Limited axonal loss, 373
 - Local anesthetic systemic toxicity (LAST), 367–370
 - in pregnancy, 213–215
 - Lorazepam, for seizures
 - from local anesthetic systemic toxicity, 368
 - Lower airway
 - obstruction, pediatric, 247–250
 - Low molecular weight heparin. See Heparin
 - Low pressure, after anesthesia induction, 376–378
 - Ludwig's angina, 30–31
- M**
- Magnesium
 - deficient, 150–151
 - for dysrhythmias in hypothermia, 156
 - for endotracheal tube ventilation difficulty, 21
 - excess, 148–149
 - for pediatric advanced life support, 271
 - for pheochromocytoma, 164
 - for thyroid storm, 168
 - Magnesium sulfate
 - for hypomagnesemia, 150
 - on neuromuscular blockers, 230
 - for seizure prevention in pre-eclampsia, 227, 228
 - for torsades de pointes/hypomagnesemia in ventricular fibrillation, 69
 - Magnetic resonance imaging emergencies, 294–296
 - Malignant hyperthermia, 158–160
 - Mannitol
 - for acute transfusion reaction, 282
 - for hypovolemia with hypernatremia, 152
 - for intracranial hypertension, 176
 - for transurethral resection of the prostate syndrome, 169
 - for traumatic brain injury, 185
 - Marburg virus, 436–437
 - Mask, N95, 57, 57f
 - Mask ventilation, difficult, 18
 - Mass casualty, 422
 - Mass casualty events, 422–423, 425, 426t
 - Mass casualty incidents, 422–423
 - Massive hemorrhage, 313–315
 - Massive transfusion, 315
 - Maxillofacial injuries, 307–309
 - McRoberts maneuver, 233
 - Mediastinal lesions, obstruction from, 108–112
 - Mediastinoscopy, major hemorrhage in, 112–114
 - Melker cricothyroidotomy, 400
 - Mental status, altered, postoperative, 330–332
 - Meperidine, for severe postoperative pain, 353
 - Metabolic acidosis, 126–128
 - Metabolic alkalosis, 131–132
 - Metabolic and endocrine emergencies, 125–171
 - acidosis, 126–128
 - adrenal insufficiency, acute, 128–130
 - alkalosis, 131–132
 - anaphylaxis, 133–135
 - diabetes insipidus, 135–137
 - diabetic ketoacidosis and hyperosmolar hyperglycemia, 137–140
 - hypercalcemia, 140–142
 - hyperkalemia, 144–146
 - hypermagnesemia, 148–149
 - hypernatremia, 151–153
 - hypocalcemia, 142–144
 - hypokalemia, 146–148
 - hypomagnesemia, 150–151
 - hyponatremia, 153–155
 - hypothermia, 155–158
 - malignant
 - hyperthermia, 158–160
 - myxedema coma, 160–162
 - pheochromocytoma, 162–164
 - porphyria, 164–167
 - thyroid storm, 167–169
 - transurethral resection of the prostate syndrome, 169–171
 - Meteorological disasters, 426–427

- Methimazole, for thyroid storm, 168
 - Methotrexate, for ectopic pregnancy, 324
 - Methylene blue, for pediatric anaphylaxis, 245
 - Methylergonovine, for uterine atony, 220, 221
 - Methylprednisolone
 - for anaphylaxis, pediatric, 246
 - for neurogenic shock, 180
 - Metoclopramide, pre-surgery, 13
 - Metoprolol
 - for atrial fibrillation, 62
 - for chest pain, postoperative, 334
 - for myocardial ischemia, postoperative, 338
 - for tachycardia, narrow complex, 66
 - for thyroid storm, 168
 - Midazolam
 - for awake intubation, 397
 - for emergency treatment, 428
 - for hypertension, postoperative, 342
 - for hypovolemia and hemodynamic instability, 85
 - for pain with burn injuries, pediatric, 254
 - for pediatric advanced life support, non-OR, 272
 - for rapid-sequence intubation, 33
 - for seizures from local anesthetic systemic toxicity, 368
 - in pregnancy, 214
 - Mitral regurgitation, 93–94
 - Mitral stenosis, 94–96
 - Morphine
 - for chest pain, postoperative, 333
 - for hypertension, postoperative, 342
 - for myocardial ischemia, postoperative, 338
 - for porphyria, 166
 - for postoperative pain, severe, 352, 353
 - Multi-drug resistant tuberculosis, respiratory protection for, 57, 57f
 - Myocardial ischemia, 81–83
 - postoperative, 337–339
 - Myxedema coma, 160–162
- N**
- N95 mask, 57, 57f
 - Naloxone
 - for altered mental status, 331
 - for hypercarbia, intraoperative, 46
 - for hypoxia, postoperative, 336
 - for pediatric advanced life support, 274
 - for respiratory depression/failure, postoperative, 351
 - for resuscitation, neonatal, 224
 - Narrow complex tachycardia, 66–67
 - Natural disasters, 426–429
 - biological, 428–429
 - geological, 427–428
 - meteorological, 426–427
 - Nausea and vomiting, postoperative, 346–347
 - NEAL, for pediatric advanced life support, 274
 - Near-drowning, pediatric, 262–264
 - Neck
 - anatomy and pathophysiology of, 316–317
 - zones of, 317
 - Neck injury, 316–318
 - Needle cricothyroidotomy, 399–400
 - Needle stick injuries, 296–298
 - Neonatal resuscitation, 223–225, 265–268
 - Neostigmine, 431
 - for postoperative hypoxia, 336
 - Nerve agents, 431–433, 432t
 - Nesiritide, for congestive heart failure, 76
 - Network failure, 383–384
 - Neurapraxia, 373
 - Neurogenic shock, 178
 - Neurologic impairment, prolonged, after regional anesthesia, 348–349
 - Neurosurgical and neurologic emergencies, 173–189
 - autonomic
 - hyperreflexia or dysreflexia, 174–175
 - intracranial
 - hypertension, 175–178
 - spinal cord injury, 178–180
 - subarachnoid
 - hemorrhage, 181–183
 - traumatic brain injuries, 184–186
 - venous air embolism, 187–189
 - Nicardipine
 - for aortic regurgitation, 89
 - for hypertension, 78
 - postoperative, 342, 343
 - for pheochromocytoma, 163
 - for subarachnoid hemorrhage, 182
 - for thoracic aortic dissection, 87

- Nifedipine, for autonomic hyperreflexia or dysreflexia, 174
- 9 Ps, 32–34
- Nitric oxide
for hypoxemia
one-lung ventilation, 116
refractory, 37, 49
for mitral regurgitation, 94
for pulmonary hypertension with amniotic fluid embolism, 201
for pulmonary thromboembolism, 55
- Nitroglycerin
for chest pain, postoperative, 333, 334
for congestive heart failure, 76
for myocardial ischemia, 82
postoperative, 338
for pulmonary edema, cardiogenic, 53
for variceal hemorrhage, 326
- Nitroprusside
for autonomic hyperreflexia or dysreflexia, 174
cyanide toxicity from, 433
for hypertension, 78
refractory or life-threatening, 78
for pheochromocytoma, 163
for thoracic aortic dissection, 87
- Norepinephrine
for acute adrenal insufficiency, 129
for anaphylaxis, 134
for laparotomy in critically ill, 310
for oliguria/acute renal failure, postoperative, 341
for sepsis, puerperal, 231–232
- Norfloxacin, for upper gastrointestinal hemorrhage, 327
- Nuclear disasters, 437
- O**
- Obstetric emergencies, 191–241
bradycardia, fetal, 205–210, 206f, 208f
breech presentation, 195–197
cardiac arrest, maternal, 216–218
coagulation factor, 198, 200, 218
dural puncture, accidental, 192–194
embolism, 198–202, 216
hemorrhage, maternal, 219–222
hypotension, 210–213
intubation, failed, 202–205
local anesthetic toxicity, 213–215
pre-eclampsia, 226–230
resuscitation, neonatal, 223–225
sepsis, 230–232
shoulder dystocia, 232–234
spinal anesthesia, total/high, 234–237
umbilical cord prolapse, 237–239
uterine rupture, 239
- Obstructive shock, 310.
See also Shock
- Occupational exposure, 296–298
- Octreotide, for variceal hemorrhage, 326
- Oliguria, postoperative, 339–341
- Ondansetron, for postoperative nausea and vomiting, 346
- One-lung ventilation, 407–409
airway pressure increased in, 117–118
hypoxemia in, 115–117
- Operating room fire, 298–300
- Opiates, for porphyria, 165
- Opioids. *See also* specific types
neonatal resuscitation with maternal exposure to, 266, 268
for pheochromocytoma, 163
for postoperative pain, severe, 352, 353
for trauma, pediatric, 279
- Oral injury, 287–290
- Organ harvesting, 443–445
- Oxygen pipeline failure, 385–387
- Oxymetazoline, for awake intubation, 397
- Oxytocin
for uterine atony, 220, 221
in uterine rupture, 241
- P**
- Pacemakers
anesthetic implications of, 394–396
after surgery, 396
before surgery, 395
in surgery, 395–396
codes of, 394, 394t
programming and modes of, 394
- Pacing
transcutaneous, 411–413
transvenous, 416–417
- Pain. *See also* specific disorders and procedures
postoperative
chest, 332–334
severe, 352–353
- Papaverine, for drug extravasation, 293
- Paraplegia, 178
- Pediatric advanced life support, non-OR, 269–274
- Pediatric emergencies, 243–280
advanced life support, pediatric non-OR, 269–274
airway obstruction, complete, 260–262

- Pediatric emergencies
(Cont.)
anaphylaxis, 244–247
asthma and
 bronchospasm,
 247–250
burns, 250–256
epiglottitis
 (supraglottitis),
 256–259, 257t
foreign body, inhaled,
 260
near-drowning,
 262–264
resuscitation, neonatal,
 265–268
stridor, postextubation,
 274–275
trauma, 276–280
Penicillin G, for anthrax, 435
Peripheral nerve injury,
 from regional
 anesthesia,
 370–373
Personal emergency
 preparedness
 plan, 424
Phenoxybenzamine,
 for autonomic
 hyperreflexia or
 dysreflexia, 175
Phenylephrine
 for awake intubation,
 397
 for bone cement
 implantation
 syndrome, 284
 for chest pain,
 postoperative, 334
 for hemoptysis, 23
 for hypertension,
 intracranial, 176
 for hypotension, 80
 with aortic stenosis,
 91
 from cardiac
 herniation, 104
 with fetal
 bradycardia, 207,
 210
 intraoperative, 169
 from major
 hemorrhage in
 mediastinoscopy,
 113
 from neurogenic
 shock, 179–180
 postoperative, 344
 in pregnancy, 211,
 212, 213
 for sepsis, puerperal,
 230–232
 for septic shock, 231
 for spinal anesthesia
 complete, 359
 total/high, 236
 for subarachnoid
 hemorrhage, 182
 for traumatic brain
 injury, 185
 for venous air
 embolism, 188
 for venous gas
 embolism, 97
Pheochromocytoma,
 162–164
Phosgene, 433
Physostigmine, for
 postoperative
 altered mental
 status, 331
Placenta accreta,
 219–222
Placental abruption,
 219–222
Placenta previa,
 219–222
Plague, 435–436
Planning, pre-disaster,
 423–425, 424t
Pneumonectomy, cardiac
 herniation after,
 103–105
Pneumothorax, 50–52,
 51f
 after neonatal
 resuscitation, 266,
 267
 from bronchopleural
 fistula, 100, 101
 chest pain from,
 postoperative,
 333, 334
 tension, 119–120
 from trauma, pediatric,
 276, 277, 278
 undiagnosed, nitrous
 oxide and, 75
Poiseuille's law, 275
Porphyria, 164–167
Porphyria cutanea tarda,
 165
Postanesthesia care unit,
 329–355
 altered mental status,
 330–332
 chest pain, 332–334
 hypertension,
 postoperative,
 341–343
 hypotension,
 postoperative,
 343–345
 hypoxia, 335–337
 myocardial ischemia,
 337–339
 nausea and vomiting,
 postoperative,
 346–347
 neurologic impairment
 after regional
 anesthesia,
 prolonged,
 348–349
 oliguria/acute renal
 failure, 339–341
 postoperative pain,
 severe, 352–353
 respiratory depression
 or failure,
 349–351
 stroke, 354–355
Postdural puncture
 headache, 194
Postoperative
 hypertension,
 341–343
Postoperative
 hypotension,
 343–345
Postoperative nausea
 and vomiting,
 346–347
Postoperative pain,
 severe, 352–353
Post-tonsillectomy
 bleeding, 13–14
Potassium
 for burn injuries,
 pediatric, 254
 excess, 144–146
 low, 146–148
Potassium chloride
 for alkalosis, 131
 for hypokalemia, 147,
 148
Potassium iodide, 437
Power failure, 379–382
Pralidoxime chloride
 (2-PAM), for
 nerve agent
 poisoning,
 432–433
Prazosin, for autonomic
 hyperreflexia or
 dysreflexia, 175
Pre-disaster planning,
 423–425, 424t
 family disaster plan in,
 423–424, 424t

- personal emergency preparedness plan in, 424
- professional emergency preparedness in, 424–425
- Pre-eclampsia, 226–230
- Pregnancy. *See also* Obstetric emergencies
 - air embolism in, 198–202
 - dural puncture in, accidental, 192–194
 - ectopic, ruptured, 322–324
 - failed intubation in, 202–205
 - heterotopic, 324
 - hypertension in, 227
 - pre-eclampsia, 226–230
 - hypotension in, 210–213
 - lidocaine with epinephrine in, test dose of, 215, 218, 237
 - local anesthetic toxicity in, 213–215
 - spinal anesthesia in, total/high, 234–237
 - thromboembolism in, 198–202
 - uterine rupture in, 239
- Preparedness, disaster, 422
- Procainamide, for tachycardia
 - narrow complex, 67
 - supraventricular, unstable pediatric, 272
 - wide complex, 70, 71
- Procedures, 393–420
 - central venous access, ultrasound-guided, 418–420
 - cricothyroidotomy, 399–401
 - cannula (needle), 399–400
 - large-bore (Melker), 400
 - special considerations, 401
- double-lumen endotracheal tube, 401–403
- femoral vein catheter, 403–405
- intubating laryngeal masks, 405–407
- intubation
 - awake, 396–398
 - retrograde, 409–411
- one-lung ventilation, 407–409
- pacemakers, anesthetic implications of, 394–396, 394t
- pacing
 - transcutaneous, 411–413
 - transvenous, 416–417
- transport, of critically ill patient, 413–415
- Prochlorperazine, for postoperative nausea and vomiting, 347
- Professional emergency preparedness, 424–425
- Progesterone, for traumatic brain injury, 186
- Promethazine, for postoperative nausea and vomiting, 347
- Propofol
 - for asthma and bronchospasm, pediatric, 248
 - for bronchospasm prevention, 39
 - for endotracheal tube ventilation difficulty, 21
 - for inhaled foreign body, pediatric, 260
 - injection pain with, 292
 - for laryngospasm, 16, 30
 - for obstructive intrathoracic/mediastinal lesion surgery, 109
 - for postoperative nausea and vomiting, 347
 - for rapid-sequence intubation, 32, 33
- for seizures from local anesthetic systemic toxicity in pregnancy, 214
- Propylthiouracil (PTU), for thyroid storm, 168
- Prostaglandin E1, for mitral regurgitation, 94
- Prostaglandin F2 α , for uterine atony, 220
- Protamine, for heparin reversal
 - with post-carotid endarterectomy bleeding, 304
 - in postoperative hemorrhage prevention, 85
- Puerperal sepsis, 230–232
- Pulmonary agents, 433
- Pulmonary edema, 52–54
- Pulmonary (thrombo) embolism, 54–56, 85–87
- Pulmonary hypertension, with amniotic fluid embolism, 201
- Pulsus paradoxus, 72

Q

Quadriplegia, 178

R

- Radiologic agents, 437
- Ranitidine, for pediatric anaphylaxis, 246
- Rapid-sequence intubation, 32–34. *See also specific disorders*
- Recombinant-activated factor VII (rFVIIa) for hemoptysis, 24
 - for massive hemorrhage, 315
- Recombinant erythropoietin, for blood transfusion refusal, 446–447
- Recovery, disaster, 422
- Red man syndrome, 133, 245
- Reflex tachycardia, 164

- Refusal of blood transfusion, 445–447
 - Regional anesthesia
 - complications, 358–373
 - complete spinal anesthesia, 358–360
 - epidural abscess, 360–362
 - epidural hematoma, 363–365
 - globe injury, 366–367
 - local anesthetic systemic toxicity, 367–370
 - neurologic impairment, prolonged, 348–349
 - peripheral nerve injury, 370–373
 - Regurgitant fraction, 90
 - Regurgitation
 - aortic, 89–90
 - mitral, 93–94
 - Relief, disaster, 422
 - Remifentanyl, for
 - obstructive intrathoracic/mediastinal lesion surgery, 109
 - Renal failure, acute
 - postoperative, 339–341
 - Respiratory acidosis, 126–128
 - Respiratory alkalosis, 131–132
 - Respiratory depression, postoperative, 349–351
 - Respiratory emergencies, 35–58
 - acute lung injury and acute respiratory distress syndrome, 36–37
 - bronchospasm, 38–39
 - ETCO₂ decreased, intraoperative, 40–41
 - hemoptysis, 44–46
 - hypercarbia, intraoperative, 46–48
 - hypoxemia, intraoperative, 48–50
 - pneumothorax, 50–52, 51f
 - pulmonary edema, 52–54
 - pulmonary embolism, 54–56, 85–87
 - respiratory
 - precautions, 57–58, 57f
 - ventilation, difficult controlled, 42–44
 - Respiratory failure, postoperative, 349–351
 - Respiratory precautions, 57–58, 57f
 - Response, disaster, 425–439. *See also* Disaster response
 - Resuscitation
 - damage-control, 280
 - neonatal, 223–225, 265–268
 - Retrograde intubation, 409–411
 - Rocuronium, for
 - rapid-sequence intubation, 32, 33
 - Rule of 9s
 - adult, 253f, 286
 - child, 252, 253f
 - Ruptured abdominal
 - aortic aneurysm, 318–321
 - Ruptured ectopic pregnancy, 322–324
- S**
- Sarin gas, 431–433, 432t
 - Scopolamine, for
 - hypovolemia and hemodynamic instability, 85
 - Sellick's maneuver, 32, 33
 - Sepsis, obstetric, 230–232
 - Severe acute respiratory syndrome (SARS), 57, 57f, 428–429
 - Sevoflurane
 - for asthma and bronchospasm, pediatric, 248
 - for endotracheal tube ventilation difficulty, 20, 21
 - for epiglottitis (supraglottitis), 258
 - Shock, 309
 - cardiogenic, 310
 - distributive, 310
 - hemorrhagic, 310, 313–315, 324
 - hypovolemic/hemorrhagic, 310
 - neurogenic, 178
 - obstructive, 310
 - spinal, 178
 - traumatic, 310
 - types of, 309–310
 - vasodilatory/distributive, 310
 - Shoulder dystocia, 232–234
 - Smallpox, 434–435
 - Smoke inhalation. *See* Burns
 - Sodium bicarbonate
 - for acute transfusion reaction, 282
 - for asystole, 60
 - for diabetic ketoacidosis and hyperosmolar hyperglycemia, 139, 140
 - for lactic acidosis in alkalosis, 131
 - for metabolic acidosis, 126–127, 160
 - for "washout acidosis" in rewarming in hypothermia, 157
 - Sodium bicitrate, for
 - aspiration prophylaxis with urgent delivery, 209
 - Sodium citrate
 - for aspiration prophylaxis in rapid-sequence intubation, 33
 - pre-surgery, 13
 - Sodium dantrolene, for malignant hyperthermia, 159, 160
 - Sodium deficit, 154
 - Sodium nitrite
 - for cyanide poisoning, 433
 - methemoglobinemia from, 256
 - Sodium nitroprusside. *See* Nitroprusside

- Sodium polystyrene sulfonate, for hyperkalemia, 145
- Sodium thiosulfate, for hydrogen cyanide poisoning
adult, 286, 433
pediatric, 254
- Spinal anesthesia
complete, 358–360
in pregnancy, total/high, 234–237
- Spinal cord injury, 178–180
- Spinal shock, 178
- Spironolactone
for congestive heart failure, 76
for metabolic alkalosis, 132
- Stenosis
aortic, 91–92
mitral, 94–96
- Stevens-Johnson syndrome, 251
- Streptomycin
for plague, 436
for tularemia, 436
- Stridor, pediatric, 275
nonperioperative onset of, 256–259, 257t
postextubation, 274–275
- Stroke, postoperative, 354–355
- Subarachnoid hemorrhage, 181–183
- Succinylcholine
after burn injuries
adult, 287
pediatric, 252
for laryngospasm, 16, 30
for obstructive intrathoracic/mediastinal lesion surgery, 110
with porphyria, 166
for rapid-sequence intubation, 32, 33
- Sufentanil, for porphyria, 166
- Sulfanegen TEA, for cyanide poisoning, 433
- Superior vena cava syndrome, 112
- Supine hypotensive syndrome, 212
- Supraglottic airway (SGA)
for cannot intubate/can ventilate, 15, 16
for endotracheal tube ventilation difficulty, 20
for mask ventilation difficulty, 18
for resuscitation, neonatal, 266
- Supraglottitis, pediatric, 256–259, 257t
- Surgery-related emergencies, 10–13
- Surgical emergencies, 301–327
abdominal aortic aneurysm, ruptured, 318–321
bleeding
after carotid endarterectomy, 302–304
after thyroid surgery, 304–306
ectopic pregnancy, ruptured, 322–324
facial trauma, 307–309
hemorrhage, massive, 313–315
laparotomy in critically ill patient, 309–312
neck injury, 316–318
upper gastrointestinal bleeding, 324–327
- Systemic inflammatory response syndrome (SIRS), 230
- T**
- Tachycardia
narrow complex, 66–67
reflex, 164
wide complex, 69–71
- Tamponade, cardiac, 72–73
from chest tube occlusion, 85
hemodynamic instability with minor blood loss from, 114
- Tenecteplase, for myocardial ischemia, 82
- Tension pneumothorax, 50–52, 51f, 119–120
- Terrorism, 431–437.
See also Intentional disasters (terrorism)
- Tetracycline
for plague, 436
for tularemia, 436
- Thermal injury. *See* Burns
- Thiocyanate, for cyanide poisoning, 433
- Thoracic aortic dissection, 87–89
- Thoracic emergencies, 99–124
bronchopleural fistula, 100–102
cardiac herniation after pneumonectomy, 103–105
hemorrhage in mediastinoscopy, major, 112–114
inhaled foreign body (adult), 105–107
intrathoracic and mediastinal lesions causing obstruction, 108–112
one-lung ventilation
airway pressure increased, 117–118
hypoxemia, 115–117
tension pneumothorax, 119–120
tracheal injury, 121–124
- Thromboembolism
in obstetrics, 198–202, 216
in pregnancy, 198–202
pulmonary, 54–56, 85–87
- Thumb sign epiglottitis, 28, 28f
- Thyroid storm, 167–169
- Thyroid surgery, bleeding after, 304
- Tissue plasminogen activator (t-PA), for postoperative stroke, 355
- TOLAC, 241

- Tonsillectomy, bleeding after, 13–14
 - Tooth avulsion, 287–290
 - Total body surface area, adult vs. child, 252, 253f
 - Tracheal extubation, high-risk, 24–27
 - Tracheal injury, 121–124
 - Tracheal obstruction, intrathoracic lesion, 108–112
 - Transcutaneous pacing, 411–413
 - Transfusion
 - blood, refusal of, 445–447
 - massive, 315
 - Transfusion reactions
 - acute, 282–283
 - delayed hemolytic, 283
 - Transport, of critically ill patient, 413–415
 - Transurethral resection of the prostate syndrome, 169–171
 - Transvenous pacing, 416–417
 - Trauma. *See also specific types*
 - cardiac, 74–75
 - dental, 287–290
 - facial, 307–309
 - pediatric, 276–280
 - Traumatic brain injuries, 184–186
 - Traumatic shock, 310. *See also Shock*
 - Trial of labor after cesarean (TOLAC), 241
 - Triple H therapy, for subarachnoid hemorrhage, 182–183
 - Triple threat model, 5–6, 5b–6b
 - Trousseau sign, 142
 - Tubal implantations, 322–324
 - Tuberculosis, multi-drug resistant, respiratory protection for, 57, 57f
 - Tularemia, 436
 - Two-person ventilation, 17
- U**
- Ultrasound-guided central venous access, 418–420
 - Umbilical cord prolapse, 237–239
 - Unfractionated heparin. *See Heparin*
 - Unintentional disasters, 429–431
 - crashes, 429
 - fires, 429–430
 - industrial, 430–431
 - Upper airway obstruction, intrinsic, 27–29, 28f
 - Upper gastrointestinal bleeding, 324–327
 - Uterine atony, 219–222
 - Uterine rupture, in pregnancy, 239
 - Uterine window, 239
- V**
- Valvular disease, 89–96
 - aortic regurgitation, 89–90
 - aortic stenosis, 91–92
 - mitral regurgitation, 93–94
 - mitral stenosis, 94–96
 - Vancomycin
 - for epidural abscess, 361
 - red man syndrome from, 133, 245
 - Vascular obstruction, from intrathoracic lesions, 108–112
 - Vasodilatory shock, 310. *See also Shock*
 - Vasopressin
 - for acute adrenal insufficiency, 129
 - for anaphylaxis
 - adult, 134
 - pediatric, 245
 - for asystole, 60
 - for hypotension, 81
 - for polyuria in diabetes insipidus, 136
 - for variceal hemorrhage, 326
 - for ventricular fibrillation, 68
 - Venous air embolism (VAE), 187–189
 - Venous gas embolism, 96–98
 - Ventilate
 - can/cannot intubate, 15–16
 - cannot/cannot intubate, 16–17
 - Ventilation
 - difficult controlled, 42–44
 - mask, difficult, 18
 - one-lung, 407–409
 - airway pressure increased in, 117–118
 - hypoxemia in, 115–117
 - two-person, 17
 - Ventilator failure, after anesthesia induction, 388–391
 - Ventricular fibrillation, 68–69
 - Verapamil, for supraventricular tachycardia, stable pediatric, 272
 - Virchow's triad, 86
- W**
- Warfarin, for post-carotid endarterectomy bleeding, 303
 - Water deficit, free, 153
 - Wide complex tachycardia, 69–71
- Y**
- Yersinia pestis*, 435–436
- Z**
- Zavanelli maneuver, 233

